



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

### Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

### About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>





MAY 1888

LANE

MEDICAL



LIBRARY

Gift





WALTER A. JILLSON. M.D.

SEP 18 1906

LANE LIBRARY. STANFORD UNIVERSITY













# DISEASES OF CHILDREN.

A TEXT-BOOK FOR THE USE OF  
STUDENTS AND PRACTITIONERS OF MEDICINE

BY

C. SIGMUND RAUE, M. D.,

CLINICAL PROFESSOR OF PÆDIATRICS, HAHNEMANN MEDICAL COLLEGE, OF PHILADELPHIA ;  
VISITING PHYSICIAN TO THE CHILDREN'S WARDS AND CHIEF OF THE CHILDREN'S  
CLINIC, HAHNEMANN HOSPITAL, PHILADELPHIA ; PÆDIATRIST TO THE  
WEST PHILADELPHIA HOMŒOPATHIC HOSPITAL ; MEMBER OF  
THE AMERICAN INSTITUTE OF HOMŒOPATHY, ETC.

---

*SECOND EDITION*

*REVISED, ENLARGED AND ILLUSTRATED*

---

PHILADELPHIA :  
BOERICKE & TAFEL.

1906.

**COPYRIGHTED**  
**BY**  
**BOERICKE & TAFEL.**  
**1906.**

**To**

**J. NICHOLAS MITCHELL, M. D.,**

**FORMERLY PROFESSOR OF OBSTETRICS IN HAHNEMANN  
MEDICAL COLLEGE, PHILADELPHIA ; IN APPRECIATION  
OF THE INSPIRATION OF HIS TEACHING, HIS  
DEEP INTEREST IN PÆDIATRICS, AND  
HIS MANY ACTS OF FRIENDSHIP  
TO THE AUTHOR**





## PREFACE TO THE FIRST EDITION

---

In presenting this work to the profession the author has aimed to make it a purely clinical one.

In the sections on treatment he has endeavored to give his own experience as much as possible, and has sought to exclude all doubtful symptoms and theoretical indications.

The section on Skin Diseases is from the pen of Dr. Leon T. Ashcraft, Lecturer on Venereal Diseases at the Hahnemann College.

In the section on Nervous Diseases, valuable suggestions have been made by Dr. Weston D. Bayley, Lecturer on Mental Diseases and Clinical Instructor in Nervous Diseases at the Hahnemann College.

C. SIGMUND RAUE.

*Philadelphia, 1899.*



## PREFACE TO THE SECOND EDITION

---

Since the appearance of the first edition of this work seven years ago, a number of important discoveries have been made in the field of Pædiatrics, and some significant changes have occurred in the views held at that time regarding the etiology and treatment of not a few of the commonest affections in childhood. Furthermore, it is but fair to say that the writer himself has felt the need for revising some of his views expressed in the former edition, for with riper years and larger experience he has learned the value of conservative methods, and has endeavored to replace the mere possibilities of therapeutics with clinical certainties.

The text has been entirely rewritten, and new matter has been added wherever it was found desirable to amplify any subject. The chapter upon Infant Feeding is practically new, and the aim has been to present in a concise and clear form the most acceptable and modern views upon this subject, which has of late years been made unnecessarily complicated. A chapter upon Diseases of the Ear, Nose and Throat has been added, and illustrations have been inserted wherever a picture or a diagram could be advantageously employed to elucidate the text.

I am again indebted to a number of my colleagues for valuable suggestions and friendly coöperation, which, to my mind, is necessary in any work covering so broad a field as that of Pædriatics. Dr. Chas. M. Thomas has kindly read the sec-

tions dealing with the diseases affecting the eyes, the ears, the nose and the throat, and has made a few additions to the manuscript. To Dr. Wm. B. Van Lennep I am indebted for assistance in revising the articles upon Appendicitis and Intussusception, and also for suggestions concerning the treatment of other conditions, wherever this has presented a surgical aspect. Dr. W. D. Bayley has kindly offered some suggestions relative to Mental and Nervous Diseases.

For the excellent index I am indebted to Dr. Ernest A. Farrington, whose painstaking arrangement of the various subjects mentioned and discussed must of necessity add to the practical value of the book. I have also to thank the publishers for their liberality in preparing the many illustrations, and for numerous other courtesies.

I cannot refrain from expressing my appreciation of the kind reception which the first edition received at the hands of the profession and of the students of our colleges, and while my aim has been not to overstep the bounds of a Text-Book, I trust that the busy practitioner will find within these pages all the practical information which he may need.

C. S. RAUE.

*1626 Walnut St., Philadelphia, Pa.*

*February, 1906.*

# TABLE OF CONTENTS

## CHAPTER I.

### HYGIENE AND NURSING.

	PAGE.
The new-born—Bathing—Clothing—The mouth and teeth—Sleep—The bowels—Airing—Exercise—Premature and delicate infants—Incubators—Therapeutic measures—Cold—Heat—Baths—Packs—Nasal syringing—Throat spraying—Inhalation—Lavage—Gavage—Irrigation of the colon—Enemata—Inunctions—Massage . . . . .	9

## CHAPTER II.

### METHODS OF CLINICAL EXAMINATION.

Periods of infancy and childhood—Diseases of infancy and childhood—Morbidity — Mortality — Growth and development—Diathesis—Temperament—Methods of taking history and keeping records—Physical diagnosis—Inspection—Palpation—Percussion—Auscultation—Pulse—Temperature—Respiration—Urine . . . . .	30
---	----

## CHAPTER III.

### THERAPEUTICS.

Stimulants—Prescribing—Dosage . . . . .	69
---	----

## CHAPTER IV.

### INFANT FEEDING.

Human milk studied in comparison with other milks and feeding mixtures—Milk analysis—Cow's milk—Causes influencing composition of breast milk—Modification of cow's milk—Other foods than milk—Weaning—Indications for varying percentages of proximate principles of infant's food—Intervals for feeding and quantity required at different ages—Sterilization of food—Pasteurizing—Preparation and indication for other foods and adjuvants to child's dietary—Artificial foods . . . . .	77
---	----

## CHAPTER V.

### DISEASES OF THE NEW-BORN.

Asphyxia—Cephaematoma—Hematoma of sterno-mastoid muscle—Intracranial hæmorrhages—Septic and other infections—Acute fatty	
--	--



	PAGE.
degeneration or Buhl's disease—Acute hæmoglobinuria or Winkel's disease—Ophthalmia neonatorum—Mastitis—Icterus neonatorum—Œdema—Gastro-intestinal hæmorrhage or melena—Gonorrhœa—Sudden death in infants . . . . .	112

CHAPTER VI.

DISEASES OF THE MOUTH.

Dentition—Abnormalities of the teeth—Stomatitis—Catarrhal stomatitis—Pityriasis linguæ—Aphthous stomatitis—Bednar's aphthæ—Aphthæ epizooticæ—Ulcerative stomatitis—Parasitic stomatitis—Gangrenous stomatitis . . . . .	124
---	-----

CHAPTER VII.

DISEASES OF THE STOMACH.

Acute gastric indigestion—Dyspepsia—Chronic gastric indigestion—Nervous dyspepsia—Acute gastritis—Chronic gastritis—Chronic gastric catarrh—Cyclic or periodic vomiting—Gastralgia—Malformations and malpositions—Contraction of the stomach—Dilatation of the stomach—Hypertrophic pyloric obstruction—Ulcer of the stomach—Cancer of the stomach . . . . .	137
--	-----

CHAPTER VIII.

DISEASES OF THE LIVER.

Jaundice—Icterus—Cholelithiasis—Acute yellow atrophy—Cirrhosis of the liver. . . . .	177
--	-----

CHAPTER IX.

DISEASES OF THE INTESTINES.

Simple diarrhœa—Acute intestinal indigestion—Acute infectious diarrhœa—Cholera infantum—Acute gastro-enteric intoxication—Acute ileo-colitis—Acute intestinal catarrh—Dysentery—Amœbic dysentery—Chronic diarrhœa—Chronic gastro-intestinal catarrh or mucous disease—Intestinal tuberculosis—Constipation—Acute intestinal obstruction—Intussusception—Appendicitis—Intestinal parasites. . . . .	183
--	-----

CHAPTER X.

DISEASES OF THE PERITONÆUM.

Acute peritonitis—Chronic peritonitis—Tuberculous peritonitis . . .	248
---	-----

## CONTENTS.

xi

PAGE.

### CHAPTER XI.

#### DISEASES OF THE RESPIRATORY TRACT.

Spasm of the glottis—Acute catarrhal laryngitis—Spasmodic croup—  
Acute bronchitis—Chronic bronchitis—Asthma—Acute broncho-  
pneumonia—Croupous pneumonia—Pleuro-pneumonia—Pulmon-  
ary tuberculosis—Pleurisy—Empyema . . . . . 254

### CHAPTER XII.

#### DISEASES OF THE HEART AND ITS MEMBRANES.

Congenital diseases and deformities—Pericarditis—Endocarditis—Myo-  
carditis—Chronic valvular disease—Mitral stenosis—Aortic steno-  
sis—Aortic regurgitation—Functional disorders . . . . . 332

### CHAPTER XIII.

#### DISEASES OF THE KIDNEYS AND URINARY TRACT.

Albuminuria—Cyclic albuminuria—Œdema without kidney lesion—  
Hæmaturia—Hæmoglobinuria—Acute nephritis—Chronic ne-  
phritis—Bright's disease—Chronic parenchymatous nephritis—  
Chronic interstitial nephritis—Diabetes insipidus—Diabetes mel-  
litus—Renal calculi—Cystitis—Enuresis—Vulvo-vaginitis—Gonor-  
rhœa . . . . . 364

### CHAPTER XIV.

#### DISEASES OF THE SKIN.

Inflammations—Eczema—Tetter—Erythema—Furunculosis, boils—  
Impetigo—Impetigo contagiosa—Urticaria, hives—Vegetable  
parasites—Tinea—Tinea tonsurans—Tinea circinata—Ringworm—  
Animal parasites—Pediculosis, lice—Scabies, itch . . . . . 393

### CHAPTER XV.

#### DISEASES OF THE BLOOD.

Anæmia—Chlorosis—Progressive pernicious anæmia—Leukæmia—  
Pseudo-leukæmia—Splenic anæmia—Hodgkin's disease—Hæmo-  
philia—Purpura . . . . . 418

### CHAPTER XVI.

#### DISEASES OF THE NERVOUS SYSTEM.

Insanity—Idiocy—Imbecility—Diseases of the brain and its mem-  
branes—Acute leptomeningitis—Tuberculous meningitis—Basilar  
meningitis—Lumbar puncture—Hydrocephalus—Convulsive affec-  
tions—Epilepsy—Tetany—Affections with motor disturbances—

	PAGE.
Chorea—Spasmus nutans—Head-nodding with nystagmus—Hysteria—Paralytic affections—Cerebral palsies—Acute anterior poliomyelitis—Infantile spinal paralysis—Family ataxia—Hereditary spastic paraplegia—Syringomyelia—Multiple cerebro-spinal sclerosis—Multiple neuritis—Symptomatic affections—Neurælgia—Headache . . . . .	438

## CHAPTER XVII.

## DISEASES OF THE EAR, NOSE AND THROAT.

Otitis—Acute catarrhal otitis media—Acute purulent otitis media—Acute tonsillitis—Acute folliculous tonsillitis—Ultero-membranous tonsillitis—Acute parenchymatous tonsillitis—Peritonsillar abscess—Hypertrophy of the tonsils—Retro-pharyngeal abscess—Acute rhinitis—Pseudo-membranous rhinitis—Simple chronic rhinitis—Purulent rhinitis—Hypertrophic rhinitis—Atrophic rhinitis—Adenoid vegetations of the naso-pharynx . . . . .	530
--	-----

## CHAPTER XVIII.

## CONSTITUTIONAL DISEASES.

Lithæmia—Uric acid diathesis—Rickets—Infantile scurvy—Barlow's disease—Status lymphaticus—Lymphatism—Scrofula—Tuberculous adenitis—Tuberculosis—Rheumatism—Acute articular rheumatism or rheumatic fever—Hereditary syphilis—Marasmus or athrepsia—Malnutrition . . . . .	567
---	-----

## CHAPTER XIX.

## ACUTE INFECTIOUS DISEASES.

Exanthemata—Measles—Rubeola—Scarlet fever—Rubella—Variola—Varioloid—Vaccinia—Varicella—Pertussis—Parotitis—Influenza—Epidemic cerebro-spinal fever—Spotted fever—Malaria—Malarial fever—Typhoid fever—Diphtheria—Membranous croup—Intubation—Glandular fever . . . . .	635
--	-----

# LIST OF ILLUSTRATIONS

FIGURE.	PAGE.
1. Apparatus for feeding premature infants, . . . . .	15
2. Infant incubator, . . . . .	16
3. Method of syringing the nose, . . . . .	20
4. Steam atomizer, . . . . .	21
5. Apparatus for performing lavage, . . . . .	22
6. Method of performing lavage, . . . . .	25
7. Weight, length, chest and head measurements under twelve months, . . . . .	33
8. Weight, length, chest and head measurements over twelve months, . . . . .	34
9. Weight chart for first month of infancy, . . . . .	35
10. Weight chart, after Holt, . . . . .	37
11. Card for recording history of case, . . . . .	42
12. Method of determining the character of a spinal deformity, . . . . .	46
13. Method of obtaining knee-jerk, . . . . .	49
14. Method of palpating the lower border of the liver, . . . . .	52
15. Method of palpating the spleen, . . . . .	53
16. Diagram showing lower border of lungs and liver, . . . . .	56
17. Diagram showing superficial and deep cardiac dulness, . . . . .	57
18. Method of holding infant during auscultation, . . . . .	59
19. Monaural stethoscope, . . . . .	60
20. Binaural stethoscope with large and small chest-pieces, . . . . .	61
21. Child of six years; lines showing percussion border of lungs, . . . . .	63
22. Anterior view, showing apex resonance, deep cardiac dulness, etc., . . . . .	64
23. Holt's apparatus for examining woman's milk, . . . . .	81
24. Diagram showing percentage of fat in whole milk and set milk, . . . . .	90
25. Diagram showing fat percentage of different layers of set milk, . . . . .	91
26. Freeman pasteurizer, . . . . .	102
27. Arnold steam sterilizer, . . . . .	103
28. Diagram showing time of eruption of the milk teeth, . . . . .	124
29. Hutchinson teeth, . . . . .	127
30. Author's acidometer for estimating acidity of gastric contents, . . . . .	144
31. <i>Oxyuris vermicularis</i> , . . . . .	243
32. <i>Ascaris lumbricoides</i> , . . . . .	244
33. <i>Tænia saginata</i> , . . . . .	245
34. Head of <i>tænia solium</i> , . . . . .	246
35. Spasmodic asthma, . . . . .	268
36. Temperature chart in lobar pneumonia, showing pseudo-crisis, . . . . .	286
37. Temperature chart in remitting pneumonia, . . . . .	287
38. Lobar pneumonia in child of four years, . . . . .	292
39. Advanced case of fibro-caseous pulmonary tuberculosis, . . . . .	306

FIGURE.	PAGE.
40. Temperature chart in empyema, developing after pneumonia, . . .	322
41. Skiagraph of child's chest, three years old, posterior aspect, . . .	333
42. Cardiac dulness at one year, six years and twelve years, . . .	334
43. Acute rheumatic endocarditis with dilatation, . . .	349
44. Acute nephritis with anasarca and ascites, . . .	371
45. Chronic parenchymatous nephritis, . . .	375
46. Method of eliciting Kernig's sign, . . .	440
47. Method of performing lumbar puncture, . . .	461
48. Hydrocephalus; early period, . . .	471
49. Cerebral diplegia, showing spastic rigidity, . . .	514
50. Climbing up the thighs in pseudo-hypertrophic paralysis, . . .	518
51. Diagram showing line of incision through the tympanum, . . .	536
52. Tonsillotome, . . .	545
53. Method of holding child for palpating pharyngeal vault, . . .	563
54. Curette for removal of adenoid vegetations, . . .	566
55. Child with rickets, showing large head, narrow chest, etc., . . .	579
56. Infant of one year with marasmus, . . .	628
57. Temperature chart in measles, . . .	639
58. Temperature chart in scarlet fever, . . .	647
59. Temperature chart in typhoid fever, . . .	705
60. O'Dwyer's set of intubation instruments, . . .	741
61. Diagram showing proper position of child in intubation, . . .	742

# DISEASES OF CHILDREN.

---

## CHAPTER I.

### HYGIENE AND NURSING.

**The New-Born.**—Although the care of the new-born belongs, strictly speaking, to the domain of obstetrics, still a few practical remarks cannot well be omitted in the introduction to the subject of nursing and hygiene of children.

As soon as the head is born the mouth and eyes should be cleansed, the latter being washed out thoroughly with a warm boric-acid solution, and this is to be followed by the instillation of a drop of a 2 per cent. solution of nitrate of silver, according to the method of Credé, if the mother be affected with a purulent or specific vaginitis.

After the cord has been dusted with powdered boric acid and dressed in sterilized cotton or gauze the child should be wiped dry, the body anointed with sweet oil, especially when there is an abundance of *vernix caseosa*, and wrapped in a warm blanket and laid aside until it is convenient to resort to the cleansing bath.

**Bathing.**—The full bath should not be given until the cord has come off, which is usually about the fifth or sixth day; stripping the cord hastens its separation. The child should be bathed always in a warm room, preferably before an open fireplace. The first bath must be a warm one, approximating the normal body temperature; in hardy children it can gradually be reduced, so that a temperature of 95° F. may be reached, by the end of the sixth month. It should be of short duration and the body dried by light rub-



bing with a soft towel. The bath is best given in the morning about one hour after feeding. In children who do not react well the full bath must be either prohibited entirely or it should be followed by a rapid sponging with alcohol and warm water, about one to two dilution. The free use of soap is a great mistake, as there is no necessity for the daily use of the same, and the irritation of the skin induced thereby often excites cutaneous eruptions.

**Clothing.**—The material should be of wool; very light weight in summer and heavier for the winter. Grosvenor (*Present Status of Pediatrics*, 1895) speaks highly of the princess-cut Gertrude suit, in which the child's organs have perfectly free play, no constricting bands being present. He also lays stress on the proper construction of the diaper, showing how the unnecessarily large, old-fashioned muslin can be the cause of much harm by overheating the buttocks and kidneys and retaining the excreta too closely. A snug fitting flannel band about the abdomen is a necessary support during infancy, as well as a safeguard against exposure and a precautionary measure in children who are prone to diarrhoea. In such children it may be worn to the end of the first dentition period, while in hardy infants it may be discarded after the first year.

**The Mouth and Teeth.**—Instead of washing the infant's mouth after each feeding it is safer to carry out the method advocated by Epstein and wash the mother's nipple with a solution of boric acid before nursing. There is more danger of carrying infection into the mouth in washing the same than by leaving it alone, and there is often much harm done to the delicate mucous membrane by rough treatment on the part of the nurse. Should thrush develop, a mild antiseptic, preferably a 2 per cent. solution of boric acid, must be used as a mouth-wash.

The care of the teeth has an important bearing on the child's health. Indigestion, enlarged tonsils, cervical adenitis, and catarrhal affections of the throat and mouth can

often be traced directly to dental caries. Beside these, there are many other conditions which show little or no signs of improvement until the dentist has been consulted, and all source of irritation from carious teeth, dental periostitis, overcrowding of the jaw, and the like, has been removed.

Much trouble can be avoided and the state of the teeth preserved in a sound, healthy condition by the daily use of the tooth-brush and early attention to the teeth showing signs of caries. There are, however, children in whom the teeth become brittle or decay in spite of all prophylaxis; such cases require constitutional treatment.

**Sleep.**—The healthy babe enjoys a peaceful, undisturbed sleep, assuming usually a graceful attitude, indicative of complete relaxation. It arouses only to take food, and is seldom awake more than one-half to one hour at a time in early infancy. After the sixth month the child gradually becomes more wide-awake during the day, requiring usually two or three naps, and about twelve hours of sleep at night, up to the age of two years. From this time on until the fourth year it should have at least one nap during the day.

Children should be carefully trained in regular habits of sleep, for if once allowed to develop, the insomnia of infants is most stubborn to overcome. The most common causes for this disorder are indigestion from over-feeding and the habit of nursing during the night, although in children of a nervous temperament it may be the result of nervous excitement induced by playing just before bedtime. Local causes, such as seat worms, must also be borne in mind.

As regards *feeding*, the first nourishment should be given at 5 A. M. and the last at 11 P. M.; rarely is it advisable to feed during the night, at least not after the fifth month, and at the age of two years the child may go without food for twelve hours during the night.

To the observant physician a sleeping infant is an interesting study, particularly so in case of illness. There are many valuable signs of disease, frequently absent during the wak-

ing state, that become prominent during sleep. As pointed out in the chapters on *Diagnosis* and *Treatment*, sleep is an important element in the recognition of diseases and in prescribing.

**The Bowels.**—The early training of the child to regular habits of stool is of the utmost importance, both from the practical and hygienic standpoint. Already in the early months of infancy the child can be taught to form the habit of emptying the bowels regularly by holding it over a small chamber, which can be held between the nurse's knees, and, if necessary, irritating the anus by means of a conical piece of soap in order to suggest the desire for stool. This should be done mornings and evenings and soon the child realizes the object of the procedure and the habit becomes established without great difficulty, perseverance and regularity on the part of the nurse being the key-note to success in obtaining such a result.

**Airing.**—The nursery should be sunny and well-ventilated, no draughts, however, being permissible. If the child is allowed to crawl on the floor, there must be a carpet in the winter, and in the summer matting can be substituted. A rug should always be at the door to prevent the draught coming through the sill and coursing along the floor, which invariably happens when the temperature of the room is higher than that of the hallway. For a similar reason it is advisable to have double windows, or at least curtains, in winter, as a current of cold air constantly flows down along the window-panes, which will surely strike the child if it be allowed to play or sleep in their vicinity.

Airing the nursery in winter is best accomplished by having the windows open in the adjoining room until the air has been perfectly purified, when the windows should be closed and the communicating door opened to allow a diffusion of the atmosphere from one room to the other. When the child can be removed from the nursery it may be aired like any other room.

In summer, the room should be kept darkened during the heat of the day ; and at sunset, when the outside air has cooled off, the windows should be opened, while the child may be taken out for an airing. Ordinarily, in the spring and fall an infant may be taken out into the fresh air at one month and even earlier during the summer. During cold weather, however, an infant under three months should not be taken out of the house, and after that age only during the sunny hours of the day. The precautions necessary to be observed in taking a child out in its coach are that it be kept out of the wind, that it be sufficiently covered and that the sun does not shine directly into its eyes, but there is no valid objection to allowing a child to sleep in the open air in clement weather providing the above precautions be taken.

Statistics show that infants require a greater amount of air-space, proportionately, than adults, and that overcrowding is a prolific source of ill-health among children. This is especially the case in institutions and hospitals for children. Infants require 1,000 cubic feet of air-space in order to thrive ; but as they grow older they develop greater resisting power to external influences, and may do well under circumstances where no more than the above, or even less, breathing-space is available for each child.

**Exercise.**—The infant gets its exercise to promote metabolism in crying and in the non-volitional movements it performs. It should, however, also have its daily sun-bath and airing and it is a good plan, once a day, to allow the infant the full and free use of its limbs by removing all tight garments and letting it lie upon a bed in this condition for a quarter of an hour. Older children require exercise of a more definite kind, such as walks in the open air, games, etc. A cold sponging every morning aids greatly in the physical development of the child. Fatigue and over-exertion in all forms of sport and exercise are to be strenuously guarded against, for the tissues and delicate organs of the growing child are far more liable to receive permanent injury

from their abuse than later in life, when they have become accustomed to accommodate themselves to the extra strains not infrequently brought upon them.

**Premature and Delicate Infants ; Incubators.**—The period of viability in a premature babe cannot be exactly stated, as the condition of the infant plays a more important role than its age. The state of nutrition at birth ; the weight and, length ; the condition of the mother during pregnancy and above all, the fact as to whether the respiratory function is active should rather decide the question of viability than a mere arbitrary age limit. The period of viability has been usually fixed at twenty-eight weeks, but a number of premature infants of twenty-four weeks have been successfully raised. Etheridge (*American Text-Book of Obstetrics*) suggests that any child that breathes at birth should be considered viable. Tarnier gives the following statistics based on five years' experience with the incubator : Infants of 6 months, 16 per cent. saved ; 6½ mos., 36 per cent. saved ; 7 mos., 50 per cent. saved ; 7½ mos., 77 per cent. saved ; 8 mos., 89 per cent. saved ; 8½ mos., 96 per cent. saved. This is fully a saving of 10 per cent. of lives, as compared with the death rate among premature infants not placed in incubators. The percentages given by Etheridge are not quite so high, but on the whole quite encouraging.

An infant weighing less than four pounds and measuring less than nineteen inches should be looked upon as premature, or at least under-developed, aside from its great feebleness and impossibility of maintaining normal body heat. These infants possess a digestive tract and respiratory organs that are imperfectly developed. The same usually holds good with the circulatory organs. Owing to the poorly developed state of the muscles, they do not have sufficient strength to suckle and deglutition is difficult. The successful rearing of these infants, therefore, resolves itself into two problems : First, the maintenance of the normal body temperature ; second, the proper mode of nourishment. Owing

to the rapid loss of heat, strength and weight incident to the early days of infancy these children must be cared for from the very beginning if we expect to save them. Infants weighing three and one-half pounds or over can usually be raised outside of an incubator. The body should be wrapped in a thick layer of cotton batting instead of attempting to dress it. In order to prevent soiling, a soft diaper is to be adjusted before wrapping up the body. The entire body is then wrapped in a blanket and hot water bags applied to the feet and sides of the body. It is well to rub the child with olive oil daily, but it should only be washed as often as is absolutely necessary.

The *diet* must be that suitable to the new-born or even more diluted (see chapter on *Infant Feeding*). If breast-milk can be obtained, this is, of course, an advantage. The milk should be taken from the breast with a pump and collected in a sterile receptacle. It is then best administered to the child with a medicine-dropper, the milk being dropped well back into the pharynx, with the child in the recumbent position. Two to four drachms of nourishment should be given hourly. If the infant cannot be made to take sufficient nourishment in this manner, *gavage* must be employed, although this procedure is less frequently indicated in these cases than in the incubator babes. I have found that partial peptonization of the food is often an advantage in premature and feeble infants owing to the under-developed state of their digestive organs. Fairchild's Peptogenic Powder answers best for this purpose; it supplies milk-sugar to the food besides a small amount of pancreatic and bicarbonate of soda.



FIG. 1. APPARATUS  
FOR FEEDING PRE-  
MATURE INFANTS.  
(KOPLIK.)

The *incubator* is a necessary apparatus for maintaining the bodily temperature in premature babes. There are a number

of patterns, each possessing good points and all differing only in minor details. The requirements of a good incubator are that it should maintain a regular degree of heat, supply the infant with a sufficient amount of fresh air and be readily accessible for purposes of changing the soiled cotton and for gavage.

The improvements and special features found in some of the more complicated incubators are advantages, but not absolute necessities.

Personally, I have had good results with an improvised incubator made out of a box with a false bottom into which hot water bottles may be placed and changed as they cool off. A thermometer must be kept in the box and the temperature watched; it should be kept at about

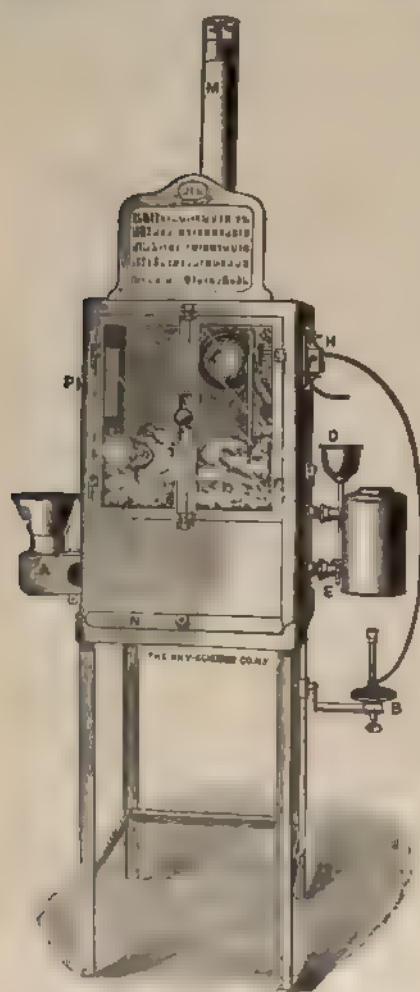


FIG. 2 —INFANT INCUBATOR

90° F. A light blanket or shawl may serve as a cover, leaving the child sufficiently exposed to allow of a free interchange of air.



At the Hahnemann Maternity Hospital Dr. Korndorfer has observed hæmorrhage into the spinal cord in two incubator babes at autopsy ; the cause of this was probably excessive heat.

*Gavage* is imperative when the babe cannot take a sufficient amount by means of the medicine dropper. One to two drachms of breast milk or of a 10 per cent. top-milk diluted five to six times with a six per cent. solution of milk sugar may be administered every hour. If the babe cannot digest plain milk, it must be peptonized.

Exhaustion is averted by handling the child as little as possible. Pads of absorbent cotton are more readily adjusted and removed than diapers, for which reason they should be used, and in place of a full bath a daily rub-down with warm olive oil is to be given. From two to four drops of brandy in twenty drops of sweetened water may be administered as a stimulant when necessary.

#### THERAPEUTIC MEASURES.

**Cold.**—In cold we have one of the best and safest antipyretics known, beside its well-known analgesic and astringent properties. To get the latter effects cold is best applied in the form of an ice-bag, a rubber coil through which ice-water is allowed to circulate, or cloths wrung out of ice-water (cold compresses). Cold is a valuable application in most inflammations, but particularly in ophthalmia, meningitis and synovitis ; as a rule, heat is preferable in inflammatory affections of the chest and abdomen. Cold compresses are useful in croup ; it is contraindicated in diphtheria (GOODNO) and in inflammations of the larynx, trachea and bronchi (HOLT).

**Heat.**—Heat is perhaps the most useful of all non-medical therapeutic measures, and has a wide field of applicability. In painful inflammatory affections it acts promptly by relieving tension and hastening resorption. The old-fashioned poultice is rapidly being superseded by hot anti-



septic fomentations in suppurative processes, which do infinitely less mischief than the former. Fomentations prepared by wringing a piece of spongiopiline or flannel out of hot water, best immersed into the same by means of a towel and wrung out by winding up both ends of the towel (the water should be slightly hotter than the hand can bear), are most serviceable when quick results are necessary, as in peritonitis, colic, etc. Dry heat is most conveniently applied by means of hot-water bags or baked flannel. It must be remembered that the child's skin is more sensitive and more readily scalded than an adult's, for which reason proper precautions must always be taken.

**Baths.**—By means of the bath we are able to apply heat or cold most rapidly to the entire body. Hot baths are often useful in collapse and asphyxia neonatorum; by adding a tablespoonful of powdered mustard to the warm bath we have an excellent means of relieving serious congestion of internal organs, through its derivative effect, and a harmless method of bringing out the rash, especially in cases of measles slow in developing.

The *bran bath* is most useful in cases of eczema or other excoriated conditions of the skin.

In cases of collapse the child may be placed in a bath of 100° F., which is gradually raised to 110°, until reaction sets in.

The action of the *cold bath* is to reduce the temperature and restore the lost tone to the cutaneous vessels, thus increasing the resistance to the blood current and improving cardiac action; besides, it gives a powerful stimulating shock to the nervous system. For this reason it is of decided value in typhoid fever.

It is best given in the following manner: The child being stripped and wrapped in a light blanket, a bath tub filled with water at 92° F. is brought beside the bed and the child immersed by means of the blanket. The temperature of the water is then reduced by the addition of cold water to 80°.

While in the bath, friction must be applied to the child's body to prevent collapse. The duration is ten minutes, and it should be repeated every three hours, reducing the temperature each time until  $75^{\circ}$  or  $70^{\circ}$  are reached, continuing at this temperature as long as the rectal temperature registers above  $103^{\circ}$  F. (BARUCH).

The cold bath is contraindicated in diphtheria and scarlet fever, and in all cases it must be remembered that the child's temperature falls more rapidly and more persistently than in the case of adults. After the bath it should be dried well and rolled up in a blanket if there is chilliness, which is seldom the case in typhoid fever, but pneumonia patients do not stand the cold so well and in these cases a gradual reduction in the temperature is always necessary as well as thorough drying after the bath.

**Packs.**—Packs are highly efficient antipyretics and diaphoretics; especially is it for the latter effect that they are employed. The *cold pack* is applied by wrapping the child in a sheet wrung out of cold water, the sheet being surrounded by a dry blanket. When used to reduce fever it can be reapplied hourly, or more frequently, as necessary. In pneumonia the pack is often restricted to the chest.

The *hot pack* is most useful in nephritis and uræmia, or suppression of urine from whatever cause. A light blanket is wrung out of hot water and applied as above, with the dry blanket on the outside.

The hot *mustard pack* is prepared by adding a little ground mustard to the hot water; it is in many instances preferable to the hot mustard bath, and is especially useful in convulsions, congestion of the lungs and of the brain; also to bring out tardy eruptions. While in the pack, the head should be sponged with cold water or water and alcohol.

**Nasal Syringing.**—This is most important in obstruction of the nasal chambers from diphtheric deposits, although cases of simple rhinitis frequently require douching to effect a prompt cure. The child is placed in the nurse's lap, its

legs held between her knees, and the arms and chest controlled by a towel; the head is inclined somewhat forward, and the blunt nozzle of a douche-bag inserted into one of the nostrils. On raising the bag, the irrigating solution flows into one nostril and out of the other, being caught in a basin held under the child's chin. The nose can also be douched with the child lying on its side (Fig. 3).

**Throat Spraying.**—The safest and most satisfactory method of bringing an antiseptic or oily solution in contact with the



FIG. 3. METHOD OF SYRINGING THE NOSE (KOPLIK.)

mucoas membrane of the pharynx and tonsils is by means of the atomizer. Children are late in learning to gargle, and even this procedure is not always to be commended, as it is at times positively harmful. In case of emergency, however, should the child be unruly, cry and not permit the use of the atomizer, it can be laid on its back across the nurse's knees, with the head thrown back, and the fluid poured into its mouth, when it will involuntarily gargle. But in employing

such a method only fluids which can be swallowed with impunity are permissible.

**Inhalation.**—The inhalation of steam is very beneficial in most respiratory ailments, but especially so in croup, and after tracheotomy it is absolutely necessary. In the absence of the specially-constructed "croup-kettle," an ordinary tea-kettle in which water is boiling may be used, the steam being directed under a sheet overhanging the child in the fashion of a tent. The steam atomizer shown in the illustration is a satisfactory instrument (Fig. 4).

**Lavage.**—The apparatus for carrying out lavage in children consists of a soft-rubber catheter, attached to a piece of rubber tubing two to three feet long by means of a piece of glass tubing, and a medium-sized glass funnel which is attached to the other extremity of the rubber tube (Fig. 5). The identical apparatus is also used for gavage. For an infant three months old I use a No. 10, English; six to nine months old, No. 11, E., and for an older infant, No. 12, K. In the new-born the catheter reaches the fundus, when introduced to the length of eight inches; in an infant of three months it must be inserted nine inches and in older infants from ten to twelve inches. I am in the habit of enlarging the eye of the catheter to facilitate the passage of mucus and curds through the same.

Stomach washing, as an adjuvant in the treatment of gastric disorders, is a procedure that has long been practiced, but its introduction into pediatric practice is due to the efforts of



FIG. 4. STEAM ATOMIZER.

Epstein, who, in 1883, published a report of 286 cases in which lavage was used in gastric disorders in infants with great benefit and without a single unfavorable result. Since then it has been extensively employed by pediatricists everywhere. Holt speaks of it as one of the most valuable therapeutic measures we possess, and he states that it has been used thousands of times under his directions without any accident whatever. While I have never seen an evil result that

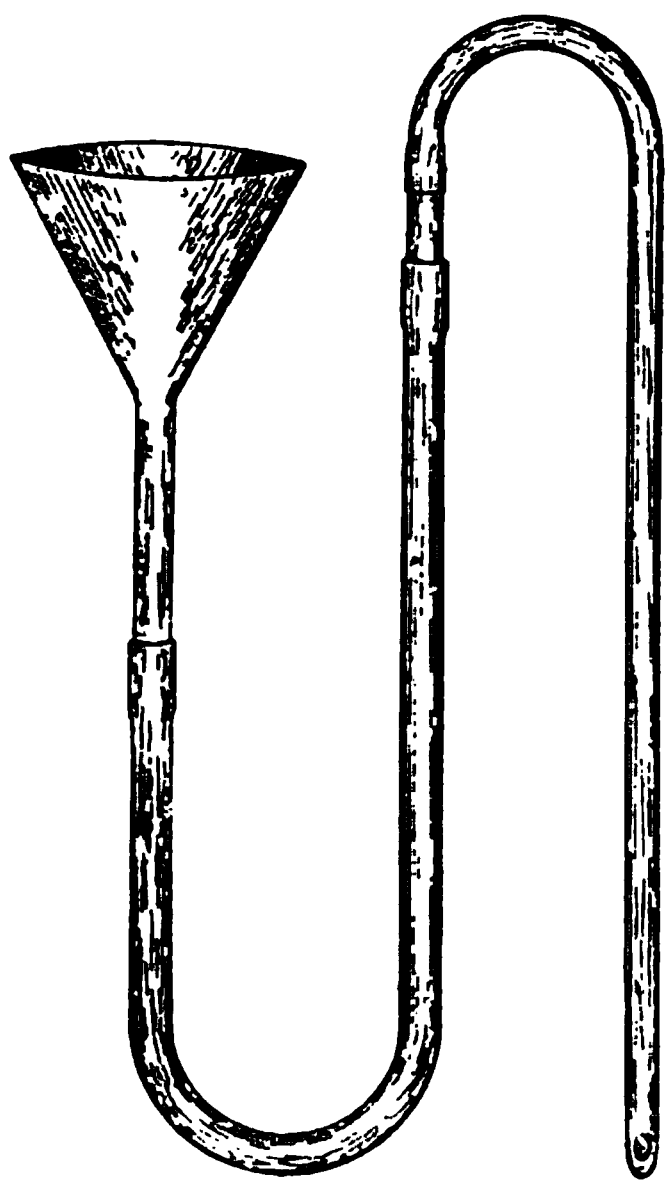


FIG. 5.—APPARATUS FOR PERFORMING LAVAGE.

could be traced directly to stomach washing, still I feel that it has its contra-indication as well as advantages, and must always be carried out with care and caution. It is hardly necessary to argue in favor of so practical and simple a procedure, and to plead for the acceptance of a mode of practice whose efforts are self-evident and whose application is based purely on the principles of common sense. We have always recognized that in toxic cases the first rule is to apply the stomach-pump. Since we have learned that most cases of acute indigestion and all cases of cholera infantum are toxic in origin, it becomes our duty

to immediately empty the stomach under these conditions unless nature has helped herself and free emesis has set in. The passage of a tube into the infant's stomach is, as a rule, accompanied by no depression and only slight discomfort, which is not to be compared to that resulting from severe nausea or artificially-induced vomiting. By this method we not only empty the stomach, but we are also able to wash

it out thoroughly and remove every vestige of harmful matter and abnormal secretions, in consequence of which, recovery from an attack of acute gastritis is more prompt than under ordinary circumstances. Besides, remedies are better able to act when taken into a clean stomach than in one containing decomposing food and mucus.

It is not, however, only in acute conditions in which lavage is of benefit; in subacute and chronic gastritis, fermentative dyspepsia and dilatation of the stomach, it has proven very useful. Daily lavage for the purpose of removing tenacious mucus that interferes with the digestive process, or for drawing off undigested food and gases where they have accumulated, is a most valuable adjuvant in the treatment of chronic gastritis and dilatation. These conditions are by no means rare, as anyone having extensive practice among children knows.

Lavage is highly recommended to allay gastric irritability and control distressing vomiting associated with obstruction of the bowels. In acute gastritis with uncontrollable vomiting there is no method of treatment so efficacious as lavage.

I wish to refer to another use to which the stomach tube may be put with great advantage, namely, for the purpose of putting food into the stomach. It may seem uncalled for to administer food in this manner, but the clinical experience upon which it is based fully justifies it. The most rebellious stomach retains several ounces of food poured in through a tube when a teaspoonful taken by the mouth will be immediately vomited. Kerley has brought this fact out prominently, and he explains it on the grounds that the passage of the tube causes less irritation of the pharynx than the food in being swallowed. Formerly, lavage was only used in the rearing of premature infants, as suggested by Tarnier, and in grave acute diseases when the child refused or was unable to take food or drink,—a condition commonly encountered in gastro-intestinal inflammation.

The results of lavage in the conditions above enumerated are positive. For the last few years I have tested it practically

both in my hospital work and private practice and my experience has led me to look upon it as indispensable in the treatment of these maladies. I have seen many cases of gastro-enteric catarrh, and some of gastric dilatation, diagnosed as marasmus (which, by the way, is a symptom, and not a disease), promptly display a tolerance for the proper food and assimilate it after the institution of systematic lavage. That it was a life saver in many of these cases I am bold to claim.

The infant, being held upright, seated on the nurse's lap, should be covered with a towel, to prevent soiling the clothing, and the catheter then inserted in the pharynx with the right hand, its tip following the index finger of the left hand, which presses down the base of the tongue (Fig. 6). Wetting the catheter with plain water is sufficient, as a rule, on account of the free secretion of mucus in the pharynx, which acts as a lubricant; but if there be abnormal dryness of the mucous membrane, there is no objection to the use of a little diluted glycerine. The child may make efforts at deglutition as soon as the catheter reaches the pharynx, in which case it glides down into the œsophagus easily. More frequently, however, it gags, interfering with the operation. If we now wait for a few seconds, until the child draws a long breath, a gentle push will readily force it into the œsophagus. All that is then required is to pass the catheter along with the fingers, which can be done without changing the position of the hand, until it reaches the stomach. This usually takes place when about ten inches have passed; and, if the stomach be full, some of its contents will escape through the apparatus when its end is lowered. In fact, the catheter can be felt to strike the fundus of the stomach, and after a little experience one can readily tell just where the tip of the catheter is located. It is well to first raise the funnel to allow the escape of gas, which is often present. It is then lowered over a basin, and held there until the stomach contents are drained off. Frequently nothing will come from the stomach until water is poured in



through the funnel and a siphon established. Again, the gastric contents may be so thick or tenacious as not to flow through the tube until diluted and broken up. With the funnel held a distance of two feet above the level of the epigastrium, two to four ounces of plain boiled water at  $100^{\circ}$  F. are poured in, and before the last part of the water has flowed



FIG. 6. METHOD OF PERFORMING LAVAGE.

in, the tube is pinched, in order to maintain a continuous column throughout the tube. The funnel is then lowered into the basin and the stomach contents siphoned out. This procedure is repeated until the fluid comes out clear.

It is often advantageous to leave a few ounces of water in the stomach; in case of vomiting, pour the feeding in before removing the tube. In acute gastritis hot water at  $110^{\circ}$  F. is



more advantageous, and when fermentation of food is a prominent symptom a 1 per cent. solution of boric acid may be used instead of plain water. I am also in the habit of using bicarbonate of soda when the gastric contents contain lactic or butyric acid. In carrying out gavage the same steps are taken, with the exception that the child is kept in the prone position throughout. The removal of the tube must be quickly done, at the same time pinching it to prevent the fluid from running into the pharynx and larynx, thus setting up gagging or a coughing paroxysm.

The contra-indications for lavage are pulmonary or cardiac diseases, with cyanosis or embarrassment of respiration, extreme debility, and ulceration of the stomach. Exceptionally, we encounter forms of gastritis in which the passage of the tube causes slight bleeding from the stomach, leading one to suspect ulceration,—post-mortem examinations, however, showing the mucous membrane intact. In such a case it is, of course, imperative to desist. Occasionally, also, we encounter an infant in which attempts at passing the tube cause much distress, embarrassed respiration, and prostration. With great care it is often possible to carry out the introduction of the tube; but it should not be long retained, and if after-effects are to be noted it is not wise to persist. The great majority of cases, however, do not mind the tube in the least, and some hardly seem to realize its presence, giving one ample opportunity to wash the stomach. Caution is, nevertheless, always necessary; and the child must be carefully watched while passing the tube, while it is in position, and after the operation.

**Gavage**, or forced feeding, is often necessary during the course of an acute illness and in certain forms of indigestion, when the child refuses to take nourishment, or is unable to do so or is unconscious. In these cases the food is introduced while the child is in the recumbent position, care being taken to keep it quiet after the operation. In cases of persistent vomiting, food introduced by means of the tube is often

retained. Premature infants are in many instances raised by gavage, when they would otherwise have succumbed without its employment.

**Irrigation of the Colon and Enemata.**—For simply emptying the rectum the enema is all that is required, but where it is deemed advisable to flush out the entire tract of large intestine it is necessary to resort to irrigation of the colon. The enema is administered by laying the child upon its back with the thighs flexed upon the abdomen and inserting the nozzle of a hard-rubber syringe, well lubricated, into the anus. Where soap and water have been decided upon for the injection, it is better to use a small fountain syringe, elevated three feet above the child's hips. The hard-rubber syringe is preferable where such substances as sweet oil or glycerin are employed. The glycerin should always be diluted with three parts water; of this, one ounce may be used.

For irrigating the colon the child is placed on its left side upon a rubber sheet covered with muslin or linen, the hips being slightly elevated. A soft-rubber catheter, No. 11 or 12, English, is attached to the nozzle of a fountain syringe, lubricated, and carefully inserted into the rectum. The water is then allowed to flow in slowly, stopping the stream as the child makes efforts at expulsion. The catheter must be passed through the sigmoid flexure as the water begins to flow and distend the rectum. The fluid will usually reach the colon without difficulty, but cannot pass into the ileum, although it is claimed that if the colon is not distended, and the water allowed to flow in slowly, a closure of the ileo-cæcal valve does not take effect, and so the fluid may even reach into the small intestines. However, if the larger bowels are thoroughly cleansed we have accomplished our end. After a half to one pint of fluid has run in, the amount being gauged according to the age of the child, it should be allowed to escape by removing the nozzle from the catheter, and the process repeated until the fluid comes out clear. Tepid water, 80° to 90° F., answers best for ordinary irrigation; boric acid may be added (2 per cent.), if desired.

Irrigation of the colon is useful in diarrhœas, especially if the stools are offensive or contain an abundance of mucus or undigested, irritating particles. The same technique is employed when distention of the bowels with water is used as a means of relieving intussusception. Hot injections have been used in collapse and cold in hyperpyrexia, but the latter procedure is entirely uncalled for. *Enteroclysis* is a safe and efficient means of averting circulatory failure in acute infectious conditions. When a hot normal saline solution is employed it stimulates the abdominal sympathetic nerves and also supplies the tissues with water. The blood pressure is promptly raised and elimination of toxins through the kidneys hastened. When the blood pressure is high and the heart chambers are over-filled it is not wise to resort to this procedure. Enteroclysis is properly carried out by inserting a Kemp's flexible double current catheter high up into the rectum and allowing water at 105° to 110° to flow through for ten to fifteen minutes. In hyperpyrexia the water should be used at 85° to 90° to bring down the temperature. It is well to leave as much water in the bowel as will remain when removing the catheter.

**Inunctions and Massage.**—The nutritive value of oil inunctions renders this form of treatment of great usefulness in all cases of malnutrition and wasting diseases. Beside the direct nutrition resulting from the absorption of the oil or fat, there is also a decided stimulus imparted to the entire nutritive process by the friction and kneading of the surface. For this reason it really encroaches upon the field of massage, from which it borrows a most useful therapeutic measure. Inunctions of an animal fat, such as benzoinated lard, not only relieve itching, but also act *antipyretically* in scarlet fever. They are valuable in any form of fever with dry, hot skin.

Massage is perhaps more limited in its field of usefulness in diseases of children when compared with its applicability in adults, but, nevertheless, there are many conditions in which it must always remain indispensable.

After the bath it is well to apply general massage to the child, especially if it be of a delicate constitution and slow to react. During the cold or graduated bath it is necessary to employ it to keep up peripheral circulation.

*Effleurage* and *Pétrissage* (stroking and kneading), together with passive motion of the joints, especially the smaller ones, are the procedures employed in the above conditions.

General massage is often of great value in cases of malnutrition, anæmia, most constitutional diseases, and especially in nervous diseases. "It becomes almost a necessary adjuvant in the functional nervous conditions in which over-feeding, combined with rest, forms the principal therapeutic means, and in organic nervous diseases generally, to promote local and general nutrition." (BARTLETT.)

Massage of the abdomen is a valuable adjuvant in the treatment of chronic constipation. The warmed hand is placed upon the abdomen in the region of the umbilicus, and under gentle pressure rotary movements are executed for a few minutes. The hand is then passed from the right iliac region upwards, following the direction of the colon, across the abdomen, and down on the left side, repeating the procedure several times. In this way friction is directly applied to the walls of the intestine, and a displacement of their contents in the normal direction is effected.

## CHAPTER II.

### THE METHODS OF CLINICAL EXAMINATION.

**The Periods of Infancy and Childhood ; Morbidity and Mortality.**—Infancy may be divided into three distinct periods, namely, the new-born, the period of early infancy and the dentition period. No sharp boundary lines can be drawn to separate these periods into distinct stages, as this classification is purely arbitrary and exists only for the sake of conveniently studying and grouping certain physiological and pathological peculiarities belonging to them.

Infancy may be said to terminate with the completion of weaning, and, although the entire teething period (twenty-four to thirty months) is sometimes spoken of as “infancy,” still the majority of pediatricists consider this terminated at the end of a year, when the child should be able to take a certain amount of solid food and plain cow’s milk. *Childhood* begins from this time on and extends up to the period of puberty (twelfth to fourteenth year in females ; fourteenth to sixteenth year in males). Childhood, again, is divided into *early childhood*, or the milk-tooth period, occupying the first to sixth year and *later childhood*, the sixth to twelfth year, during which time most of the permanent teeth erupt and physical and physiological processes more closely attain to the adult type.

**The Diseases of Infancy and Childhood.**—While in many instances it is correct and permissible to speak of *diseases of children*, still a large number of diseases encountered in childhood are but the ordinary ailments that affect all mankind in general. Their course, however, is so modified by the immature or exaggerated anatomical structure and physiological activity of the child’s economy that they differ in many respects from the type of the disease as seen in adults. Croup-

ous pneumonia, typhoid fever, enteritis, etc., belong to this group. Capillary bronchitis, spasmodic croup, the exanthemata and a number of other contagious diseases belong almost exclusively to the period of childhood, while rickets and hereditary syphilis are distinctly diseases of children.

The new-born is particularly susceptible to septic infection on account of the open state of the umbilicus and the delicate nature of the epidermis. Besides, there are distinct pathological conditions belonging to this period. They are spoken of as the diseases and malformations of the new-born (*Neonatorum*).

The young infant is particularly susceptible to mycotic disease of the mouth (*thrush*) owing to the absence of normal buccal secretion. It may also develop capillary bronchitis or contract whooping cough or succumb as a result of congenital debility, hereditary syphilis or early tuberculous infection.

The teething period predisposes to gastro-intestinal derangements, although in this period of infancy a large number of infants succumb to broncho-pneumonia. Disturbances of nutrition belong to this period—marasmus, rickets, scurvy.

Childhood proper gives us the largest number of acute infectious diseases. The intermingling of children on the street and at school explains the prevalence of contagious disease at this period of life.

**Mortality.**—Nearly 10 per cent. of all infants die during the first month of life (ERÖSS). From a study of the death reports of New York City, Holt found that about one-fourth of all deaths occur during the first year of life and nearly one-third during the first two years. The causes for this high mortality are mainly congenital debility, improper feeding and the infections.

The largest number of deaths occurs from gastro-intestinal diseases, which are most fatal in the hot summer months. They furnish about 35 per cent. of deaths. Next come the

acute diseases of the respiratory tract, 21 per cent. Other prominent fatal diseases are whooping cough, 12 per cent.; congenital syphilis, 10 per cent.; measles, 9 per cent. (ASHBY and WRIGHT.)

**Growth and Development.**—The rate of increase in the infant's *weight* is a safe criterion for judging of its progress, while continued loss in weight possesses distinct diagnostic significance. Absence of the regular weekly gain in weight implies improper feeding providing there are no signs of disease present. When not directly traceable to insufficient nourishment or indigestion we should suspect the advent of marasmus, or the beginning of a tuberculous meningitis, or general infantile tuberculosis.

Progressive increase in weight cannot, however, be looked upon as an invariably favorable sign. It is well known that syphilitic infants often look fat and well nourished, but may, nevertheless, die very unexpectedly. Budin (*Annales du Med. et de Chir.*, June, 1900) has observed that infants suffering from various acute disorders may gain in weight suddenly and then die in the course of a few days. In some of these cases there is localized œdema and deficient urinary excretion. In febrile disturbances he has also noted increase in weight at times.

Hand in hand with increase in weight there should also be a regular increase in length in the normally developing infant. According to Schmid-Monnard there is an increase in length of three-quarters of an inch per month during the first year. The male new-born measures 50 c.m. in length; the female, 49 c.m.

During the first two months of life there is a gain of from 3 to 4 c.m.; in the following three months, 2 c.m.; and in the last months of the first year, 1.5 c.m. At the end of the first year the total gain is 19 to 23 c.m.; at the end of the second, 10 c.m., and during the third year, 7 to 8 c.m. The male slightly exceeds the female in length (MONTI).

The head has a greater circumference than the chest at

birth ; at the middle of the first year the measurements begin to approximate each other and at the end of the year the chest grows larger than the head. A comparison of the circumference of the head with that of the chest, therefore, offers important clinical data. In rickets the head is some-

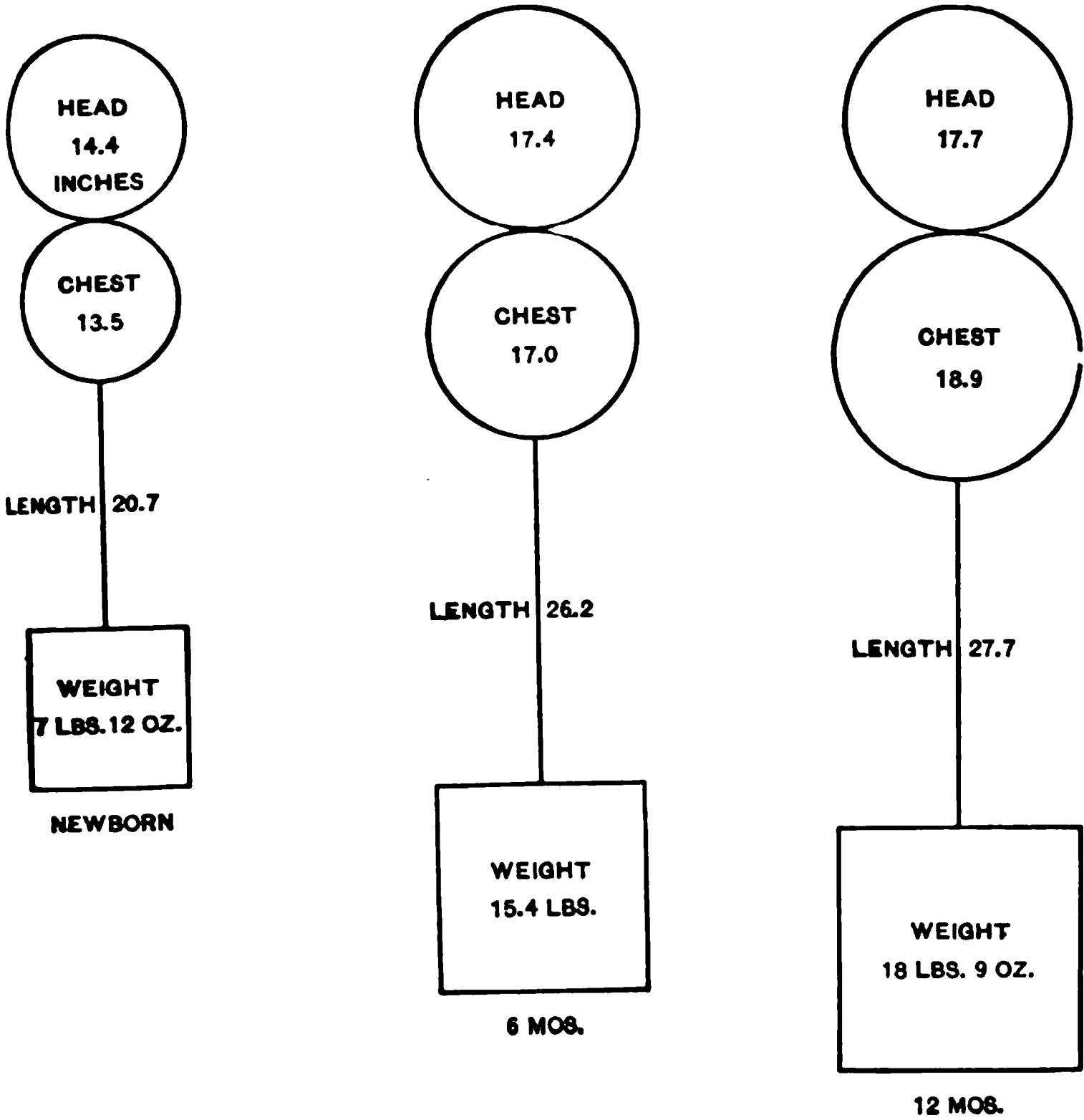


FIG. 7.

what larger than normal while the chest is abnormally small. In hydrocephalus the head is unusually large and the chest normal, while in microcephalus the head is proportionately much smaller than normal.

The diagrams shown in the illustrations (Figs. 7 and 8) have been constructed from the results of measurements of 200



healthy infants by Hedlicka and Pisek (Chapin's *Theory and Practice of Infant Feeding*, pp. 306 and 307).

The *initial weight*, roughly stated, may be said to double

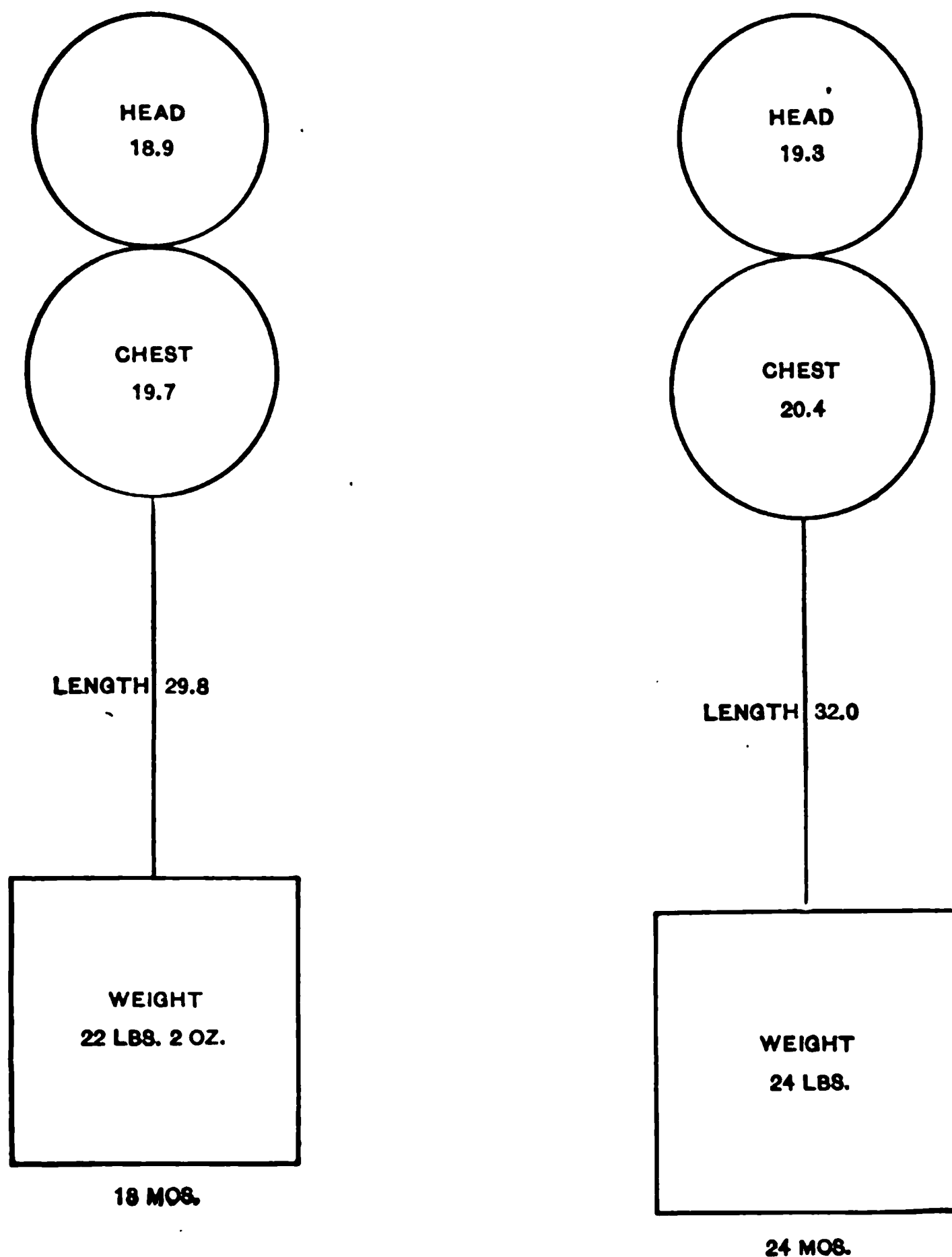


FIG. 8.

itself in five months and treble itself at the end of the first year. During the first four months the babe gains half a pound per week ; this gradually falls off until there is from

one-half to one-third of that amount of weekly gain. The average infant weighs seven and one-half pounds at birth, losing half a pound during the first week. Not until the

Revised No.

## HAHNEMANN HOSPITAL—Maternity Department

## WEIGHT CHART

NAME	Color	Sex	Date of Birth	190
DAY	W	EA		
MONTH	DAY			
1	2	3	4	5
6	7	8	9	10
11	12	13	14	15
16	17	18	19	20
21	22	23	24	25
26	27	28	29	30
31				
32				
33				
34				
35				
36				
37				
38				
39				
40				
41				
42				
43				
44				
45				
46				
47				
48				
49				
50				
51				
52				
53				
54				
55				
56				
57				
58				
59				
60				
61				
62				
63				
64				
65				
66				
67				
68				
69				
70				
71				
72				
73				
74				
75				
76				
77				
78				
79				
80				
81				
82				
83				
84				
85				
86				
87				
88				
89				
90				
91				
92				
93				
94				
95				
96				
97				
98				
99				
100				
101				
102				
103				
104				
105				
106				
107				
108				
109				
110				
111				
112				
113				
114				
115				
116				
117				
118				
119				
120				
121				
122				
123				
124				
125				
126				
127				
128				
129				
130				
131				
132				
133				
134				
135				
136				
137				
138				
139				
140				
141				
142				
143				
144				
145				
146				
147				
148				
149				
150				
151				
152				
153				
154				
155				
156				
157				
158				
159				
160				
161				
162				
163				
164				
165				
166				
167				
168				
169				
170				
171				
172				
173				
174				
175				
176				
177				
178				
179				
180				
181				
182				
183				
184				
185				
186				
187				
188				
189				
190				
191				
192				
193				
194				
195				
196				
197				
198				
199				
200				
201				
202				
203				
204				
205				
206				
207				
208				
209				
210				
211				
212				
213				
214				
215				
216				
217				
218				
219				
220				
221				
222				
223				
224				
225				
226				
227				
228				
229				
230				
231				
232				
233				
234				
235				
236				
237				
238				
239				
240				
241				
242				
243				
244				
245				
246				
247				
248				
249				
250				
251				
252				
253				
254				
255				
256				
257				
258				
259				
260				
261				
262				
263				
264				
265				
266				
267				
268				
269				
270				
271				
272				
273				
274				
275				
276				
277				
278				
279				
280				
281				
282				
283				
284				
285				
286				
287				
288				
289				
290				
291				
292				
293				
294				
295				
296				
297				
298				
299				
300				
301				
302				
303				
304				
305				
306				
307				
308				
309				
310				
311				
312				
313				
314				
315				
316				
317				
318				
319				
320				
321				
322				
323				
324				
325				
326				
327				
328				
329				
330				
331				
332				
333				
334				
335				
336				
337				
338				
339				
340				
341				
342				
343				
344				
345				
346				
347				
348				
349				
350				
351				
352				
353				
354				
355				
356				
357				
358				
359				
360				
361				
362				
363				
364				
365				
366				
367				
368				
369				
370				
371				
372				
373				
374				
375				
376				
377				
378				
379				
380				
381				
382				
383				
384				
385				
386				
387				
388				
389				
390				
391				
392				
393				
394				
395				
396				
397				
398				
399				
400				
401				
402				
403				
404				
405				
406				
407				
408				
409				
410				
411				
412				
413				
414				
415				
416				
417				
418				
419				
420				
421				
422				
423				
424				

average normal babe not to regain its birth-weight until after ten days. The accompanying chart is used at that institution for recording the daily weight (Fig. 9).

Holt has constructed a weight chart (This chart is published by Geo. L. Goodman & Co., 55 Fulton St., New York City, and has on its reverse side spaces for recording the diet from month to month, Fig. 10), which is indispensable for accurately recording the weight and obtaining the *weight curve*. The normal weight curve is indicated on the chart and by this we can gauge the progress of the case. In older children the average weight at the various ages is about as follows :

AGE.	HEIGHT.	WEIGHT.
Birth	20 in.	7 ½ lbs.
5 mos.	24 in.	15 lbs.
1 year	29 in.	21 lbs.
2 yrs.	32 in.	26 lbs.
3 yrs.	35 in.	31 lbs.
4 yrs.	38 in.	35 lbs.
5 yrs.	40 in.	40 lbs.
6 yrs.	43 in.	44 lbs.
7 yrs.	45 in.	48 lbs.
8 yrs.	47 in.	53 lbs.
14 yrs.	60 in.	100 lbs.

The *fontanels* offer positive indication of the progress of development. Normally the posterior fontanel is obliterated at the end of the second month, while the anterior closes from the sixteenth to the eighteenth month. Ordinarily, delayed closure of the anterior fontanel indicates malnutrition or rickets. When bulging is associated with separation of the cranial sutures the cause is more likely to be hydrocephalus.

*Muscular development.* At three to four months the babe attempts to grasp objects and can hold up its head. By the seventh month it should be able to sit erect, and before it is a year old it should make voluntary efforts to support the weight of the body with its legs, *i. e.*, stand with slight assistance. Walking is as a rule attempted at the end of a year. The ability to walk unassisted should not be delayed beyond

The first Legend here represents the average major axis of a bounding legend during the first run and is based upon 24.000 observations by a computer (Table 2).

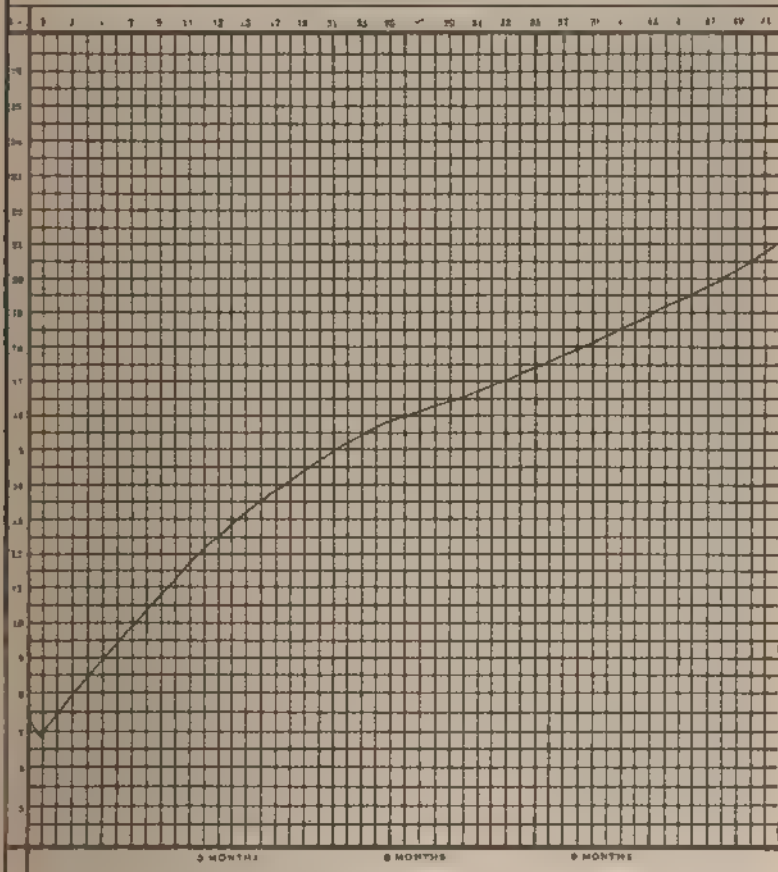
### INFANT'S WEIGHT CHART.

Name \_\_\_\_\_

[illegible]

Date of Birth

WEEK OF AGE.



the eighteenth month. If so, malnutrition or rickets should be suspected.

*Talking* begins coincidently with walking. As a rule, girls begin earlier than boys. At the age of two years a child should be able to put words together intelligently. Aside from tongue-tie—a rare condition—the causes delaying the development of speech are either constitutional enfeeblement or some mental defect (see chapter on Nervous and Mental Diseases).

The physiological and anatomical peculiarities distinctive of the period of childhood will be discussed in the introductory remarks to the chapters dealing with the various organs and systems.

**Diathesis ; Temperament.**—Much stress was formerly laid on the value of the diathesis as an important element in diagnosis, and teachers of pediatrics attempted to classify the various constitutions into definite types, each of which showed pronounced predisposition to certain diseases. It was held that the scrofulous diathesis, for example, was a distinct form of constitution in which there was a tendency to suppurating affections of the lymphatics certain skin diseases, predisposition to catarrhal affections, croup and meningitis, and a number of other constitutional disturbances, among which enlarged tonsils and adenoid vegetations stood prominently. The condition was not clearly understood until it was discovered that scrofula, so-called, was in reality tuberculosis of the lymphatics. A tuberculous diathesis was also spoken of, in which there was a strong predisposition to pulmonary tuberculosis and other acute forms of the disease. We know now that tuberculosis may affect any child that has been exposed thereto, as many authenticated cases demonstrate ; and, while there is no doubt that certain individuals are more susceptible to the tubercle bacillus than others, still the so-called tuberculous diathesis is nothing more than a frailty of constitution, and has no other significance.

It is improper to speak of a syphilitic diathesis, as is some-

times done, because a person either has syphilis or has it not, there being no proof that such a thing as natural predisposition, any more marked than the universal predisposition of all mankind, exists. On the other hand, if syphilis be inherited, the patient is immune to acquired syphilis.

The rheumatic diathesis has been described, but there is still confusion in the minds of the profession as to what is really meant by this term. To the writer's mind, this diathesis occupies at present the position formerly occupied by the scrofulous diathesis, which we now fully understand. Speaking of the hereditability of rheumatism, Bartlett says: "The hereditability of rheumatism is universally conceded. And yet the present popular view that it is an infectious disease will probably do much to modify this opinion. The difficulties can be reconciled by assuming that there is a special diathesis favoring the incidence of rheumatism, and that this is transmitted from parent to child. It is also suggested that the poison upon which rheumatism depends is the special agency which is transmitted. Still others assert that it is some particular anatomical or structural peculiarity which is responsible." (*A Text-Book of Clinical Medicine.*)

It will therefore be seen that it is unsafe, in the present state of our knowledge of diathetic conditions, to place too much importance on this feature of a case, or to go beyond the teachings of Bouchard, who divides constitutions into the *arthritic*, or a predisposition to certain diseases in which the process of nutrition is retarded (rheumatism, gout, diabetes, cholelithiasis, etc.), and *scrofula*, in which there is a predisposition to tuberculosis. The writer has faithfully endeavored to demonstrate to his entire satisfaction that the study and recognition of the diathesis is an important clinical datum, but he has not been convinced that this is always the case. (*Hahn. Monthly*, Feb., 1903.)

The following description of the various diatheses is given for the sake of acquainting the student with the characteristics held to be *sui generis* of these types of constitution.

The *scrofulous* child is stout and flabby, and is subject to glandular enlargements and catarrhal conditions of the mucous membranes and skin; the features are usually coarse, the temperament phlegmatic, and the cerebral faculties dull.

The *tuberculous* or *phthisical* child is of an active temperament, bright and precocious; the frame is sparsely developed, the skin delicately transparent, the hair generally soft and silken.

The *syphilitic* infant is recognized by the hoarse cry, the snuffles, ulcerated nasal septum, the characteristic eruptions, especially in the groins and about the anus, and the old, withered look, due to malnutrition. Later in life we notice the broad, flat root of the nose, the linear scars about the angles of the mouth, Hutchinson's teeth, interstitial keratitis, and many other possibilities. Of course, it is unreasonable to expect to find all of these signs in every case of hereditary syphilis, but careful examination will usually detect sufficient of them to clinch the diagnosis.

The *rachitic* child is typical in appearance. When well developed there is the characteristic square head, the epiphyseal enlargements, the beading of the ribs, bowing of the long bones, pot-belly, enlarged spleen, profuse sweat about the head, anæmia and constipation. In abdominal tuberculosis we also have the large belly; but here the small chest, the wasted thighs and the absence of typical rachitic manifestations will easily differentiate the two.

*Rheumatism* is more extensive in its areas of distribution in children than in adults. The joints are not, as a rule, so severely affected as the endocardium and nervous system. Tonsillitis, with fever and aching in the limbs, is often the only outward manifestation of an acute attack of genuine rheumatism, during the course of which the heart is often involved, or chorea follows as a sequel. The rheumatic diathesis, therefore, often presents itself by nothing more than the common joint-pains, often called growing pains; urinary disturbances, pointing to incomplete oxidation and elimina-

tion of excreta; a general retardation of the nutritive processes, from which gravel and biliary calculi may result; anæmia; subcutaneous fibrous nodules; chorea and endocarditis, and certain forms of cutaneous eruptions.

#### METHODS OF TAKING A HISTORY AND KEEPING RECORDS.

The importance of intelligent, systematic case-taking and the keeping of accurate records cannot be overestimated. By using the card system the physician can keep a single set of records, including both his office and outside work, for it is a simple matter to carry a few cards in one's visiting list, and thus take notes at the bedside of the patient.

The first part of the history comprises the data obtained by interrogating the child or the attendant upon the child. After this has been recorded the results of the physical examination and such remarks as the physician finds of direct bearing on the case (prognosis, diagnosis, treatment) are added thereto.

The schema shown in Fig. 11 is a reproduction of the card used in the children's department of the Hahnemann Hospital Dispensary. The significance of the data sought and the means by which they are best obtained are as follows:

The *family history* is inquired into for the purpose of determining whether there is an hereditary disease or hereditary predisposition. Sometimes, as in the case of tuberculosis, it is difficult to say whether heredity or exposure to a tuberculous relative plays the most prominent part in the case. Inquiry should be made regarding tuberculosis in parents or their immediate relatives. A history of syphilis can at times be obtained by a frank admission of the parents, but often they will not only deny the same but even evade skillfully applied interrogations aimed at establishing such a history. Rheumatism should also be inquired into, as there is no doubt that certain diseases are based upon a special diathesis favoring the incidence of rheumatism (see *ante*). *Atavism*, the ten-



dency of certain diseases, notably tuberculosis, to reappear after having skipped a generation, must also be taken into consideration. Mental and nervous diseases in the parents or immediate relatives should be noted. Temperamental peculiarities and neurotic tendencies may be augmented in the child through the intermarriage of near relatives.

The health of the other children and their number may throw much light upon the family history. Marasmus commonly appears when the mother has had a large number of

No. ....		<b>CHILDREN'S DEPARTMENT</b>	
<b>HAHNEMANN HOSPITAL DISPENSARY.</b>			
Name		Date	
Address		Nativity	
Age		Occupation	
Diagnosis			
HISTORY:			
Health of father			
"    " mother			
"    " other children			
Mode of birth		Dentition	
Food			
Previous illnesses			
Present environment			
Present illness			

FIG. II.

children in close succession. A history of *miscarriages*, together with stillborn children or the death of preceding children in the early months of infancy from "inanition," point strongly to maternal syphilis.

The *mode of birth* may account for the presence of birth palsies (protracted labor, especially breech cases).

It is of prime importance to learn whether the child was

*breast fed* or *artificially fed*. Improper artificial feeding is the cause of the various nutritional diseases, such as rickets, marasmus, scurvy and gastro-intestinal catarrh. It may also be the origin of tuberculosis. Unsuitable breast-milk and prolonged lactation, however, not infrequently bring on rickets. Note what the present food is.

Note the time at which the *teeth* made their appearance and whether dentition progressed steadily or with interruptions; also the time of *walking* and *talking*. Dentition, walking and the state of the *fontanel*s are indices of the physical development of the child, while talking is an important index of mental development.

*Previous illnesses.* Has the child had the various infectious diseases? Name them to the mother individually. As most of them occur only once in a lifetime, a doubt in the diagnosis is at once removed if the child has already had the disease we may be suspecting. Vaccination must not be forgotten. Aside from the question of *immunity* we must also consider that certain diseases have sequelæ or predispose to other diseases. Thus, measles and whooping cough predispose to tuberculosis; scarlet fever may leave nephritis or chronic suppurative otitis, and the latter may be the cause of some obscure intracranial condition (cerebral abscess, thrombosis). Convulsions in infancy may terminate in epilepsy in later childhood. Diphtheria may be followed by paralyses of various kinds.

*Environment.* Aside from offering a source of infection, environment may affect the child's constitution to a marked degree. The squalid, sunless tenement houses furnish ample cause for anæmia and rickets, even tuberculosis. Children raised in the country rarely develop rickets. Overwork at school or an exacting teacher may be the etiologic factor in chorea. Again, many vicious habits are directly attributable to environment.

The *present illness* is now recorded. Inquire into the child's health before the first signs of the ailment showed

themselves. Determine whether there were prodromata and whether the disease developed slowly or abruptly. Exactly how many days has the child been ill? If there is fever, since when, and has the fever been continuously high, remitting, or intermitting? What other important symptoms are present—vomiting, diarrhoea, constipation, cough, pain? If there is pain, where does the child refer the pain to, and is it aggravated by motion? Has the child complained of *sore throat*?

In describing the *stools*, inquire into their size and frequency, color, odor. Is mucus, blood or undigested food-matter present? Is there pain before, during or after the stool?

Having completed the interrogation of the case, the findings of the physical examination according to the methods detailed below are then added in clear and concise terms, together with the findings in the urine, sputum and blood when called for.

For facilitating the recording of physical signs rubber stamps giving the outlines of the front and posterior aspect of the trunk are very convenient. The diagram is simply stamped upon the record card, and by means of lines and arbitrary signs to indicate the outlines of organs and pathological findings a graphic representation of the case is obtained for future reference. These stamps can be obtained at most surgical instrument houses.

#### PHYSICAL DIAGNOSIS.

**Inspection** is the first step in the examination of a sick child. What has been discussed in the previous section should be put to practical application in beginning the study of a case, and so the diathesis, temperament, state of development and nutrition, and individual peculiarities of the patient are first to be noted. If the child be of the tuberculous diathesis, presenting the constitution and temperament peculiar to the same, we naturally suspect the possibility of pulmonary

mischief; or, if such a child complains of a pain in the knee, we immediately turn our attention to the hip-joint rather than consider the pain to be of rheumatic origin, in which case we would expect to find other prominent evidences of rheumatism.

To inspect the child satisfactorily it must be stripped and viewed from front and back, both standing and reclining.

The *diathesis* having been noted, the development of the *framework* should next demand our attention. Is the child emaciated? If so, in what particular locality is this most marked? The prominent belly, small chest and wasted thighs have been referred to.

The color of the *skin* is important to note. Normally, in the infant it is pink, and anæmia is not difficult to recognize. Eruptions must be looked for, eczema and syphilis being the most common conditions encountered at this period of life. In cardiac and pulmonary disease, and especially in membranous croup, *cyanosis* is to be observed. *Jaundice* also is a condition often seen in the new-born and is not foreign to childhood. Miliaria and sudamina are common in rachitic children, especially in summer. By drawing the finger-nail across the skin a red streak will be left, indicating an instability in the vaso-motor nerves. It is very pronounced in disease of the central nervous system, for which reason Trousseau attempted to establish this symptom pathognomonic of meningitis. The phenomenon is known as *tache cerebrale*.

If there be deformity of the *spine*, we must determine whether it is due to Pott's disease, rickets, a unilateral pleural effusion, old pleuritic adhesions, or lack of muscular development. The child should be laid flat upon its stomach and the body then partly lifted from the table by making traction on the feet. If rachitic, the deformity is at once reduced by the traction, but the kyphosis of Pott's disease is irreducible under all methods of manipulation (Fig. 12). Retraction of the chest from pleuritic adhesions produces scoliosis, and in these cases we can get the history of a former empyema as well as confirmatory physical signs in the thorax.

The *head* presents many peculiar features of prominent diagnostic value. In rickets it is large and square; in hydrocephalus large but rounded, the fontanelles are widely open, and the eyeballs displaced downwards. In rickets there are often parchment-like areas representing a thinning out of the bony elements, known as *craniotabes*. The osseous nodes of syphilis are very characteristic.



FIG. 12 - METHOD OF DETERMINING THE CHARACTER OF A SPINAL DEFORMITY

The *facial expression* often points to the seat of trouble; for instance, the knitting of the brows in headache, which when associated with squinting is a strong presumptive sign of meningitis, the fan-like motion of the alæ nasi in respiratory troubles, and the pinched expression of the face in abdominal disease. Roughly speaking, it can be said that the upper part of the face represents cerebral, the mid-portion respira-

tory, and the lower portion abdominal disturbances. Often one cheek will present a circumscribed redness, which is said to correspond to the side affected in pneumonia. Personally I have seen it change from side to side. In severe pulmonary infiltration or congestion and in some forms of heart disease the obstruction to the circulation will become manifest by networks of enlarged capillary vessels seen on the cheeks (also on the chest, and sometimes on the palms of the hands).

The *chest* may present deformities, peculiarities of the ribs, deviations from the normal respiratory movements, abnormal movements, and various skin eruptions. In the early stages of pleurisy the painful side becomes fixed and may produce a certain degree of scoliosis. As the effusion is poured out the side bulges. In chronic pleurisy with adhesions the side becomes permanently retracted. The *intercostal phonation phenomenon* of Stiller (*Wiener Med. Wochenschr.*, No. 15, 1902) is a bulging or elevation of the lower intercostal spaces seen when the patient is made to enunciate sharply words of short syllable. It is due to a wave of air propagated down the bronchial tree as the air is being forced through the narrowed glottis. This wave is transmitted to fluid effusions in the pleural sack, but the sign is absent when pulmonary consolidation is present.

In rickets the sternum is prominent from lateral compression of the costal cartilages (*pectus carinatum*), and the pathognomonic beading of the ribs, the "rickety rosary," is often present. In phthisis that portion of the chest over the consolidated lobe is flattened and does not move in the same degree as the unaffected side; the clavicle stands out prominently, and there is often marked retraction of the ribs (flattening) in that region.

In emphysema the chest assumes a rounded fullness, slight motion only being perceptible during respiration. After pericarditis with adhesions the intercostal space is often seen to retract distinctly during the heart's diastole, but more important than this is *Broadbent's sign*, i. e., systolic retraction of the lower ribs posteriorly on the left side.

The *spine* has been referred to. Spina bifida must not be overlooked.

The *limbs* and *joints* must be examined for evidences of arthritis or tuberculous joint troubles; the fibrous subcutaneous nodules pathognomonic of rheumatism; the deformities of rickets, rheumatism, and poliomyelitis anterior; the bone affections of syphilis and tuberculosis. The limbs will also give evidence of the various forms of paralysis likely to occur in childhood, and of rachitic pseudo-paralysis.

The *reflexes*. Among the superficial reflexes the plantar is of especial importance. Under normal conditions a flexor response is obtained, but in lesions of the pyramidal system hyperextension of the great toe occurs. This is spoken of as *Babinski's sign*. In infants up to the age of learning to walk the response is somewhat similar to the Babinski phenomenon. The great toe is drawn back; the toes are extended and spread out and the foot is everted. The Babinski sign is more deliberate, however, and there is but a small amount of movement at the ankle.

The *knee-jerk* is exaggerated in lesions affecting the upper neurons or irritating the lower neurons. Diminished or abolished knee-jerk indicates lesions in the lower neurons. In children it is best obtained in the dorsal position with the foot resting on the palm of the left hand, striking the tendon with a percussion hammer held in the right hand (Fig. 13).

*Ankle clonus* indicates disease in the spinal cord, from the first to third sacral segments.

The *position* assumed by the child during sleep and waking is important to note. We see the child burying its head in the pillow in cerebral inflammations; lying on the back with limbs drawn up in abdominal inflammations; on the affected side in acute pleurisy; the head drawn back and the spine arched during opisthotonos; unable to lie in the prone position in the dyspnoea of capillary bronchitis; impossibility of extending the leg upon the thigh when in the sitting posture owing to contraction of the flexor muscles, which disap-



pears when the dorsal decubitus is assumed (*Kernig's sign* in meningitis); sleeping or comatose; crying out in sleep and gritting the teeth. During natural sleep the child assumes an easy, graceful position, indicating complete relaxation; the respiration is of the abdominal type.

The character of the *cry* is often a hint in diagnosis. The shrill, piercing cry of meningitis is pathognomonic. The hoarse cry heard in the absence of croup points to syphilis. In otitis the cry is often continuous, in spite of all that is

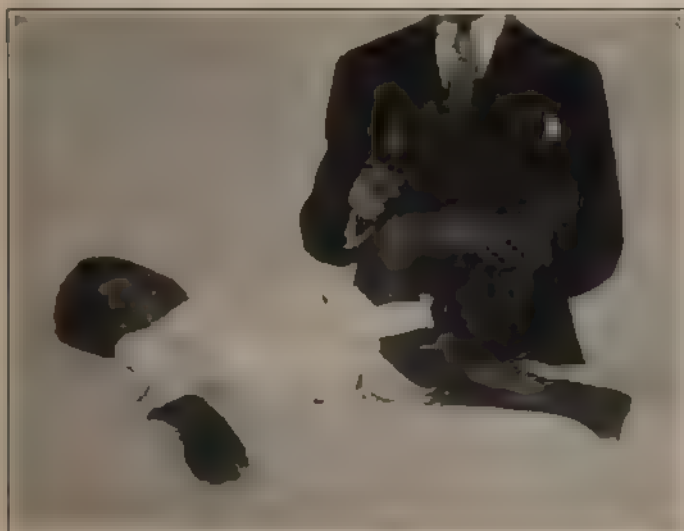


FIG. 13. —METHOD OF OBTAINING KNEE-JERK.

done to humor the child. The recognition of the cry of hunger, pain and temper is more readily attained by observation than from reading. The natural cry is a loud, strong vocal effort accompanied by reddening of the face and does not last more than a few minutes. Abnormal cries are as a rule weaker in character and more persistent. The cry of pain may be strong, but it is accompanied by evidences of suffering and distress, such as facial contortions, drawing up of the legs, bringing the hand to the affected part, etc., and it is more or



less persistent. The cry of hunger is a continuous fretful cry, ceasing when food is offered. The cry of temper is loud and is accompanied by all the signs of anger, such as kicking and striking about. During serious illness the cry becomes feeble and partakes more of the nature of fretting.

The inspection of the *throat* is left until the last on account of the struggles and resistance of the child usually induced thereby. It should be done quickly and thoroughly, and all preparations relative to the examination must be made beforehand. Taking the child into a good light, or in some cases a head mirror may be used, it is seated upon the nurse's lap or held in her arms, the head slightly thrown back, and the handle of a spoon pressed down firmly on the base of the tongue. This is very often followed by gagging or a violent expulsive effort, but if we are quick we have seen enough. The gagging brings to view every part of the fauces, it being desirable to gain access to the lateral regions. In contagious diseases we should be prepared for the sudden cough which is likely to occur and spurt mucus or pieces of membrane into our face. Often nothing more will be necessary than to allow the child to cry, during which act a satisfactory view of the mouth and throat is obtainable. We must cultivate the habit of taking in the whole picture at a glance and retaining the impression long enough to analyze it, otherwise much valuable time will be spent in bungling efforts.

**Palpation.**—The sense of touch, when properly trained, will give more information in the study of sick children than is generally supposed. The first thing that strikes our attention as we touch the child's body is the temperature, and with a little practice this method of judging of the degree of fever becomes accurate enough for many cases. We should observe whether the heat is uniform, or whether one part of the body is hotter than another, for the head may be considerably hotter than other portions of the body; in the later stages of entero-colitis the abdomen will be hot, while the extremities may be decidedly cold.

In palpating the *head* we determine the state of the fontanelles, whether they be delayed in closing or prematurely closed, whether bulging or depressed. We also look for craniotabes, exostoses, and any evidence of sensitiveness of the scalp or ears, this often hinting at middle-ear disease when other prominent signs are wanting. If this sensitiveness to touch be general, it marks the advent of rickets (JENNER).

From the head we can descend to the *chest*, taking in the *neck* on our way down, where we often find scrofulous enlargement of the cervical glands. Often, however, no definite sequence can be followed out, and we must avail ourselves of an opportunity presented by the child either crying, ceasing to cry, or finding it in a sound sleep, to proceed at once to palpate the abdomen, which can only be done satisfactorily during complete relaxation.

In an examination of the chest palpation is usually the first step, and if the child will accommodate us by crying we can judge of the vocal fremitus. The child should be held by the mother in such a manner that it rests on one of her shoulders and presents its back to the physician. The hand is placed on the back in order to determine vocal fremitus, and the rattling of mucus in the bronchi is distinctly transmitted to the hands, in bronchitis. The hands can then be placed on the sides of the chest and the respiratory movements of both sides compared. The left hand will now seek the cardiac area, by which means hypertrophy or a thrill can often be detected.

Auscultation should follow next in chest examinations, for the disturbance induced by percussion may be so great as to hinder any further progress in the case.

The *abdomen* is most satisfactorily palpated while the child is asleep, the warmed hand being gently introduced under the bed-covering. Distension or retraction of the abdominal wall was noted while inspecting. The trained palpating hand will recognize enlargement of the liver and spleen (Figs. 14

and 15); the presence of enlarged mesenteric glands; friction between the abdominal wall and the organs, impacted fecas, etc.

Tenderness in certain regions and rigidity of the recti muscles is of diagnostic significance. Thus, tenderness over McBurney's point and rigidity of the right rectus is pathognomonic of appendicitis. Gurgling in the right iliac fossa together with tenderness is strong presumptive evidence of typhoid fever, but not a pathognomonic sign.



FIG. 14.—METHOD OF PALPATING THE LOWER BORDER OF THE LIVER IN THIS CASE THE LIVER WAS SLIGHTLY ENLARGED.

The bladder may be felt in the hypogastrium when distended, and in rachitic children with flabby abdomen it is often possible to palpate the kidneys. A *rectal examination* should be made as a supplement to the abdominal examination in all doubtful cases.

The *thighs* offer a valuable indication of the state of nutrition. If the adductor muscles are wasted, soft and flaccid to the touch, and the skin capable of being pinched up into folds, slow to disappear, we have a marked picture of wasting.

The *skin* furnishes valuable diagnostic signs. The tem-

perature has been noted. The state of dryness or moisture is determined by palpation; often an eruption can be better felt than seen, and the shotty feel of the skin in the early stages of small-pox is very characteristic.

The *tache cérébrale* has been referred to. It is a hyperæmic streak obtained by irritating the skin in cases of meningitis—a patch of angio-paralytic area.

**Percussion.**—The usual order of examination in adults cannot be observed in children, as has been already pointed out. On account of the disturbance it is likely to produce in the



FIG. 15.—METHOD OF PALPATING THE SPLEEN.

child's tranquility, percussion is best left to the last in chest affections, just as inspection of the throat is put off until all other data have been obtained, when the disease points to that locality.

Percussion of the *head* is of little value excepting for the purpose of eliciting tenderness, especially over the mastoid region, when ear disease is suspected. *Macewen's sign* is a hollow note elicited by percussing over the anterior part of the skull and is indicative of distention of the lateral ventricles with fluid. It is found in meningitis and is sometimes a valuable early sign.

In percussing the chest of the child we must bear in mind that owing to the lesser dimensions of the thorax and the greater elasticity of its walls it becomes more difficult to outline the organs and to demonstrate the differences in the intensity and pitch of the percussion note at various points. The explanation of this phenomenon lies in the fact that the percussion impulse is transferred over a greater area than in the adult on account of the resilience of the thorax. It is, therefore, necessary to percuss more lightly, not only for fear of eliciting deep dulness from adjacent organs not directly under investigation, but also because it is impossible to outline the superficial dulness by strong percussion. This applies especially to the heart and thymus gland, although the same holds good in percussing the abdomen with the object of outlining the lower border of the liver or an enlarged spleen, etc.

Again, it is easier to judge between the presence or absence of resonance in a certain locality than to estimate differences in pitch and intensity. Light percussion alone makes this possible.

In percussing out a superficial organ (thymus) or a superficial area of dulness of an organ situated like the heart (the "absolute dulness") the best results are obtained by pressing the middle finger of the left hand lightly against the chest wall and striking quick, gentle taps with the middle finger of the right hand. When striving to elicit deep dulness in order to outline a deep-seated organ like the spleen or determine the deep ("relative") dulness of the heart or liver, the finger must be pressed more firmly against the chest and the percussion strokes dealt more strongly, avoiding, loud percussion, however, which drowns out the finer shades of distinction between the notes and practically abolishes all border lines. In percussing, the examiner's finger also experiences varying degrees of *resistance*, which is a great aid in locating the boundaries sought for and in recognizing the physical nature of pathological processes capable of impairing resonance.

The elicitation of deep dulness is handicapped by certain sources of error, and the results are often misleading. In the first place, there is greater likelihood of our percussion strokes not being of uniform force, and it is difficult to determine just how energetic they should be in order to outline the extent of space through which an organ can diminish the normal resonance of the thorax. Secondly, the area obtained may exceed in size the actual size of the organ under examination. This is especially so with the heart, as Sahli has demonstrated (*Die Topographische Percussion im Kindesalter*, Bern, 1882).

The factors influencing the percussion note over the lungs, independent of the adjacent organs, are, according to Sahli:

(a.) The thickness of the thoracic wall. It is an established fact that the percussion note obtained over the lungs under equally strong percussion is the more intense the thinner the wall is that covers the area percussed.

(b.) The configuration of the thorax plays an important rôle in the difference in the intensity of the resonance in different localities. Convexity of the thoracic wall tends to diminish the intensity of the percussion note, as a greater part of the percussion impulse is required to depress the convex wall sufficiently against the underlying structures to set them into vibration than is the case with a plane or concave thoracic wall. For this reason the flattened areas of a rachitic thorax give an apparent hyper-resonance when compared with areas of normal configuration. This modifying factor must also be borne in mind when percussing chests deformed by scoliosis and kyphosis.

(c.) The close apposition of the ribs in a certain region will give rise to dulness at that point. This is often seen in cases of pleurisy before exudation has set in, and may give rise to a diagnostic error. The explanation of the displacement of the normal relationship between the ribs is a voluntary scoliosis from fixation excited by the pain in the affected side (WERNER).

(d.) The percussion note obtained in an intercostal space is

of greater intensity than that obtained over a rib. This is of practical importance in outlining the heart, as apparent dullness beginning at the second rib may be due to the rib and not to the underlying upper border of the heart, as percussion in the second intercostal space will prove.

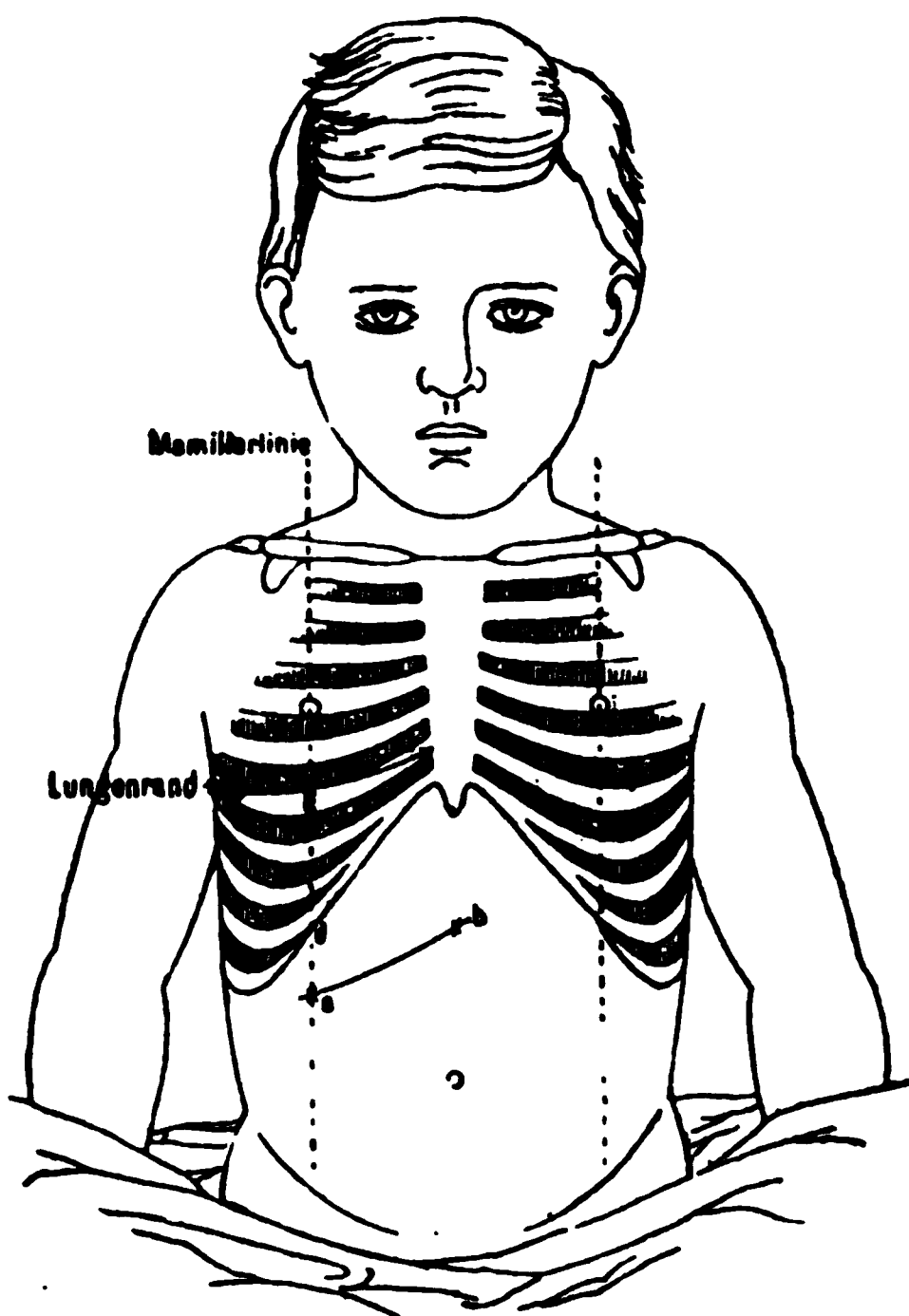


FIG. 16.—DIAGRAM SHOWING LOWER BORDER OF THE LUNGS AND LIVER. (SAHLI.)

Normally, the percussion note gradually increases in intensity both anteriorly and posteriorly as we descend, and then gradually diminishes as the lower border of the thorax is reached. The increase in intensity in percussing downward results from greater thinness of the thoracic wall—the pectoral muscles and the scapula and its muscles padding the upper part of the thorax considerably—and the flatter configuration of the chest at its mid-portion. As we descend we impinge



upon the deep dulness of the liver and spleen posteriorly and the liver and heart anteriorly.

The *lower border of the lungs* in the dorsal position is identical in children and adults, and not higher as Weil claimed SAHLI). The following points reach the extreme lower border of the lungs: Right mammary line, upper border of sixth rib; left mid-axillary line, upper border of ninth rib;

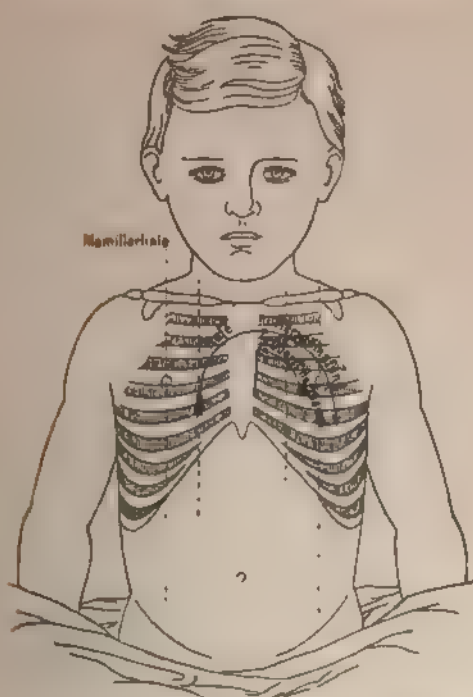


FIG. 17.—DIAGRAM SHOWING SUPERFICIAL AND DEEP CARDIAC DULNESS. (SAHLI.)

posteriorly, on either side of the spine, eleventh dorsal spine, (Fig. 16).

The percussion note over the *sternum* is more intense than in the adult owing to the elasticity of the thorax, for which reason the percussion stroke is carried to a greater part of the lungs than merely the underlying portion. A slight shade of



difference naturally exists and is apparent when percussing from an adjacent region of the thorax toward the sternum and over it, but it is no more pronounced in degree than the difference existing in the note over a rib and in an intercostal space. Percussion of the sternum in children, therefore, gives more positive results than in the adult. In percussing from above downwards the upper boundary of the deep cardiac dulness may be traced, providing we do not use too strong a stroke (Fig. 17). The presence of the *thymus gland* may also be demonstrated in the upper sternal region in young children. In the lower sternal region cardiac dulness is demonstrable. Jacobi recommends percussion of the sternum from below, the child being supported face downward, when any difficulty is experienced in outlining the thymus.

Normally the child's thorax is hyper-resonant in comparison with that of the adult, and owing to the pliability of the chest walls a cracked-pot sound can often be elicited, especially when the child is crying. The possibility of emphysema and cavity existing must, however, not be forgotten.

The posture during percussion is important. If the child does not sit perfectly erect and the spine is curved so as to bring the ribs closer together on one side than on the other, we will obtain dulness over this area. Likewise when percussing the back, dulness may be elicited where it should in reality not exist if the mother holds the child tightly against her chest in presenting the child's back to us for percussion. Again, dulness due to a pleural effusion changes its level with a change in the position of the child. Crying also causes dulness in the bases posteriorly, owing to the prolonged expiratory effort.

In *abdominal* disease percussion is of great value. The abdomen may be distended either from gas, fluid, or solid growths, and percussion, together with the signs of fluctuation, when obtainable, will make a differential diagnosis possible.

The boundaries of the liver and spleen can be percussed out satisfactorily with sufficient practice.

**Auscultation.**—In auscultating the chest of a young child it is most advantageous that it be held by the mother as shown in Fig. 18 with its back exposed to the examiner. This is the position in which the back and lateral regions also can be most satisfactorily percussed. When we wish to auscultate anteriorly the child is put into the crib on its back.



FIG. 18. METHOD OF HOLDING INFANT DURING AUSCULTATION.

The proper time to auscultate is when the child happens to be in a tranquil mood, and as a rule it is wise to begin the examination with auscultation. Should it then begin to cry, we make use of the crying sounds to determine the vocal resonance and the presence or absence of bronchophony.

Older children may be engaged in conversation when we wish to study the voice sounds.

There are two methods of auscultation, namely, the immediate and the mediate. In immediate auscultation the ear is placed directly upon the chest—it being preferable always to interpose a towel between the physician's ear and the patient's body—while in mediate auscultation the *stethoscope* is used to convey the sounds from the bare chest to the examiner's ear.

The beginner should first master immediate auscultation and after he has learned to recognize the various sounds and interpret them he may avail himself of the comforts and advantages of the stethoscope. Even the skilled examiner finds it advantageous first to listen with the naked ear and then more accurately localize certain sounds and verify his findings by means of the stethoscope. Deep-seated lesions in the chest may be overlooked when the stethoscope alone is used, for the naked ear is able to perceive sounds originating at some depth below normal lung tissue which the stethoscope fails to transmit.

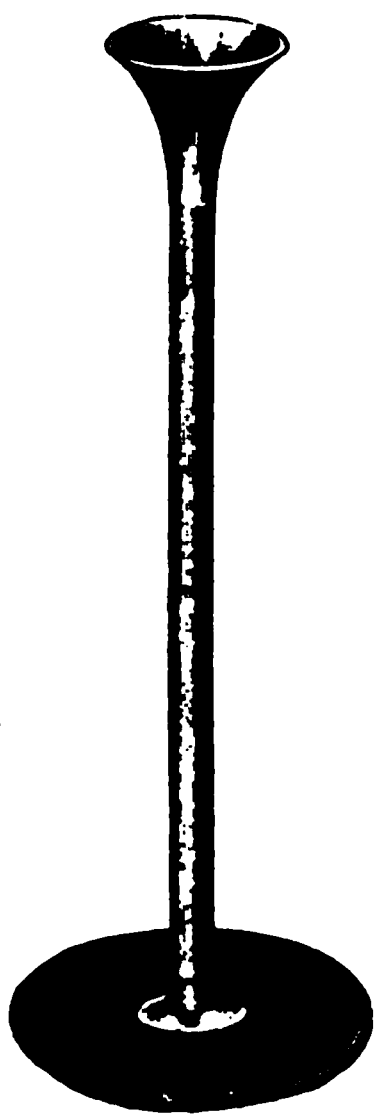


FIG. 19.—MONAURAL STETHOSCOPE.

The main scientific purpose of the stethoscope is to ascertain and isolate the sounds from small, circumscribed areas, as the chestpiece of the stethoscope does not conduct sounds from so wide an area as does the ear. There is no doubt that the *monaural* instrument (Fig. 19) is the more desirable one for this purpose, but from the standpoint of practicability the *binaural* surpasses it, especially when working with children. With this instrument it makes no difference whether the child be restless or quiet or whether it be too sick to be taken up and held properly—it is always possible to get at the chest without putting oneself into an uncomfortable position.

The disadvantages of the binaural stethoscope are that it produces extraneous noises, and while it magnifies low pitched sounds it does not convey certain feeble, high pitched sounds as clearly as the naked ear or the monaural instrument: With constant practice, however, and by checking one's findings by immediate auscultation whenever possible, these disadvantages can be overcome entirely.

Instruments for magnifying sounds, such as the phonendoscope and the Bolles' stethoscope, while at times convenient, are on the whole objectionable. True, one can listen through the clothes with them and examine a patient without even turning him, but such practice is not to be encouraged. As Sahli says (*Klinische Untersuchungs-Methoden*) the difficulty encountered in auscultation lies not in hearing the sounds in the chest but in interpreting their meaning.

The stethoscope shown in Fig. 20 has two sizes of chest-pieces which is a feature at times most desirable. The thumb-piece gives one a good hold on the instrument without danger of touching the tubing and producing extraneous sounds. The ear-pieces must be adjusted to each individual.

The *heart* can be auscultated posteriorly almost as well as anteriorly in infants and the murmurs of congenital heart disease are often better heard between the scapulæ than over

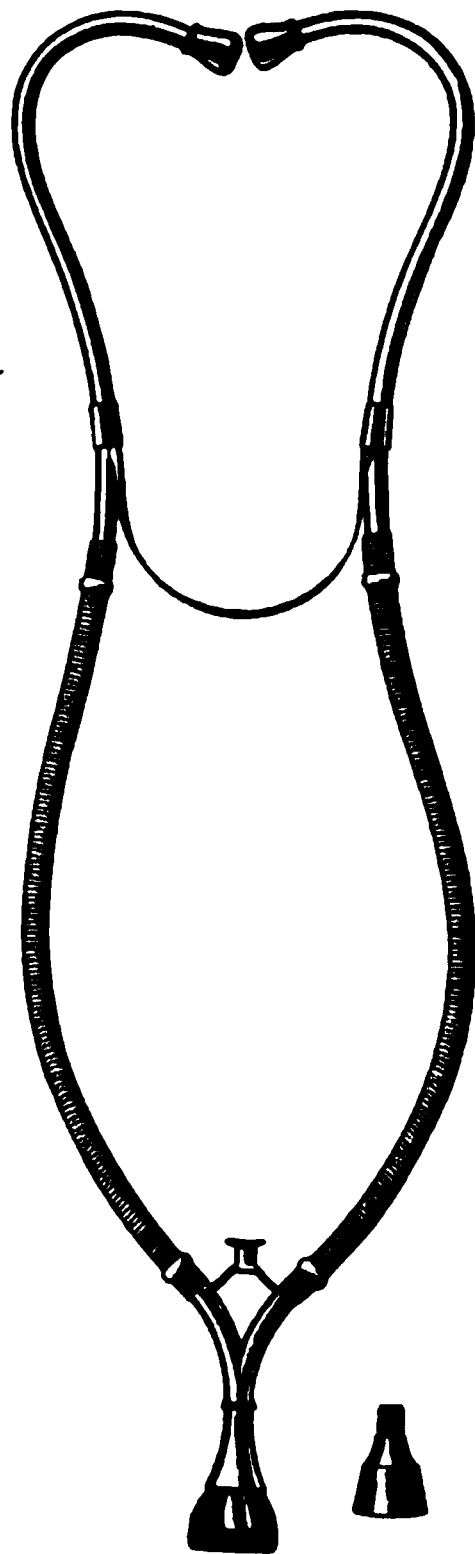


FIG. 20.—BINAURAL STETHOSCOPE WITH LARGE AND SMALL CHEST-PIECE.

the cardiac area in front. The heart should always be auscultated as a routine practice. This should be the first step, as crying makes it impossible to discover anything abnormal.

Normally the first sound at the apex is the loudest sound of the heart. Next in intensity is the pulmonary second, and lastly the aortic second sound. The rhythm is trochaic (Hochsinger). The pulmonary second may be equal to or even louder than the aortic up to the time of puberty (tenth to twelfth year). The explanation of this difference between the sounds of the child's heart and that of the adult lies in the fact that the arterial tension is much lower—the ratio between the volume of the left ventricle and the aorta being less than in adults—while the pressure in the pulmonary circuit is higher.

Auscultation is seldom of use in abdominal conditions in children, excepting to determine the absence of intestinal movements as occurs in diffuse peritonitis.

In auscultating the *lungs*, crying does not interfere unless associated with a harsh laryngeal note. In other ways it is an aid in giving us a deep inspiration and an audible expiration.

The respiration of the child is of the *puerile type*, characterized by a harsh, sonorous inspiration somewhat bronchial in character. This type of breathing is encountered in the adult when the vesicular breathing becomes exaggerated or increased in intensity by extraneous causes compelling the lung or a part of it to assume increased activity. In this change both the inspiratory and expiratory factors are proportionately increased in loudness and in length, inspiration however being more accentuated than expiration (Tyson). In the infant, owing to the slight movements of the chest wall and the purely abdominal mode of breathing, the respiratory sounds are feeble.

As the right bronchus is of larger calibre than the left, the respiratory note is more intense on the right side. Bronchial breathing may be heard to the right of the spine in the scap-

ular region, or to be more exact, broncho-vesicular breathing (Fig. 21). If the diaphragm be forced up by gaseous distention of the abdomen the vesicular murmur will be suppressed at the bases of the lungs.

Occasionally during deep inspiration, especially during crying, sub-crepitant râles may be heard at the apices (supra-clavicular region) and at the bases posteriorly. In pneumonic



FIG. 21. CHILD SIX YEARS OLD. LINES SHOWING EXTREME PERCUSSION BORDER OF LUNGS AND INTERLOBULAR FISSURES. BRONCHIAL BREATHING HEARD AT (O).

conditions we must be on our guard not to confound the harsh, rasping *sub-crepitant râles*, characteristic in children, with pleuritic friction sounds and diagnose pleurisy where it does not exist.

**Pulse, Temperature, Respiration.**—As in adults, the pulse is best felt at the wrist in children, although it can at times be estimated with advantage through the anterior fontanelle.

The pulse is very rapid in infants, gradually decreasing in frequency during childhood, attaining the average rate of 76 in males and 80 in females by the time of puberty. In young children the rhythm is variable and irregular, owing to the incomplete development of the physiological inhibitory centres. The pulse-rate is often affected by physiological influences to such an extent that it cannot be taken as a safe criterion of



FIG. 22.—ANTERIOR VIEW OF CHILD SHOWN IN FIG. 21.  
APEX RESONANCE, DEEP CARDIAC DULNESS AND  
LOWER BORDER OF LUNGS OUTLINED

fever, which can only be surely determined by palpation and thermometry.

During the first weeks of life the pulse-rate varies between 125 and 150 beats per minute; more rapid in female infants, as a rule, and not influenced by posture.

From the sixth to the twelfth month it is usually 105 to 115, and more susceptible to bodily exercise.

From the second to the sixth year it may be said to vary within 90 to 105 beats; seventh to tenth year 80 to 90 beats, after which it gradually attains the average adult standard.

The strength of the pulse is our guide in judging of the heart's condition, and must be carefully observed during the course of the acute infectious fevers and in pulmonary inflammations.

One of the most satisfactory results to be obtained in studying the pulse is when we compare it with the temperature and respiratory ratio. Thus in the beginning of typhoid fever the temperature may have risen several degrees above normal while the pulse-rate is still unaffected. Later it may rise entirely out of proportion to the temperature. The pulse does not, therefore, rise in a uniform ratio with the rise of temperature in all cases, although as a rule one degree of fever-heat is usually accompanied by an increase of eight pulse-beats.

"A pulse-rate rather slow in proportion to the temperature is favorable, indicating a tranquil nervous system. A low pulse with high temperature invites us to look for spinal cause, as pressure on the brain or depressing action of drugs. A low temperature and frequent pulse points to local complications in the thorax or pelvis (WUNDERLICH).

"A slowness in the pulse has often a great significance in the diagnosis of cerebral affections, and especially meningitis" (FINLAYSON).

Irregularity in the pulse is also found in meningitis, combined with slowness. When the pulse is more rapid, the fever prominent and the breathing embarrassed, we should suspect peri- or endocarditis.

"The number of respirations per minute does not correspond so closely to the temperature as the frequency of the pulse. In collapse there is often (not always) a frequency of respiration, and in slight fever of childhood also; in moderate fever the respirations amount to 20 or 30 per minute; in children to 40 or 50. In considerable or extreme degrees of



fever they are higher yet, 60 in many cases ; movement also increases their frequency." In pneumonia and congestion of the lungs the rate of respiration is entirely out of proportion to the fever and pulse, and greatly quickened respirations should at once lead us to examine the chest.

The temperature is best taken by inserting a clinical thermometer, lubricated with vaselin, into the rectum. It is usually a trifle higher than in the mouth, but it is much more satisfactorily taken here, and far more accurately than in the axilla or groin. The diurnal variation in the temperature is more pronounced than in adults, varying within a range of from two to three degrees. The lowest temperature is attained shortly after midnight, when it may be as low as 97° F. in the rectum, rising to a height of 100° F. in the afternoon, in some instances.

**The Urine.**—The difficulty of obtaining a specimen of urine for chemical examination, and of estimating the total quantity in twenty-four hours, leaves the clinical study of urine in infancy and childhood a much-neglected branch. Fortunately the necessity for studying the urine does not arise as frequently in children as in adults, but when presenting itself it is of the highest importance that we should know how to proceed. For ordinary purposes a clean sponge can be placed over the genitals and held in place by the diaper, which should have a layer of oiled silk or other impervious material on its inner surface. When the child has micturated, the urine is squeezed from the sponge into a clean vessel. By measuring the quantity thus obtained and noting the number of urinations in twenty-four hours we can quite accurately estimate the total quantity. Often the variations in the frequency of urination are a safe enough guide in estimating the functions of the kidneys. Should the quantity obtained by the method detailed above not be sufficient for a chemical examination, the process can be repeated until enough is obtained. Instances may arise where resort to the catheter will become necessary, in which case a sterilized No. 5 to 6 soft-rubber catheter is to

be employed. The urine can sometimes be forced from the bladder by gentle stroking in the suprapubic region, it being received into a beaker glass held under the penis. Simply irritating the prepuce will often excite urination.

The *daily quantity* of urine gradually increases from an ounce at birth to six to ten ounces by the end of the second week. The amount is relatively large during early infancy, increasing from six to twelve ounces at the first month to sixteen ounces at the sixth month. By the second year it may reach twenty ounces, and by the eighth year two pints and over. The specific gravity is relatively low during infancy, the percentage of solids being far below that of adolescence, but the amount of urine passed is greater in comparison with the body-weight than in adults. The frequency of urination gradually decreases as the child develops and gains more control over the sphincter vesicæ; the act is involuntary until after the second year.

The variability of the character of the urine in childhood is well known. At times it will be high-colored, staining the napkin, and causing the child to cry while urinating, on account of the presence of urates and uric acid; again, it may be turbid from mucus or phosphates, especially the latter in intestinal indigestion. The odor is in many cases quite pronounced, from the presence of aromatic compounds.

*Albumin* should immediately be suspected when the urine imparts a slight amount of stiffness to the diaper on drying; in fact, it may be so abundant as to stiffen the cloth like starch. It is normally found in the urine of the newborn. Blood is most likely to originate in the kidneys in childhood, especially in scarlatinal nephritis, and give the urine a smoky appearance. Hæmaturia in infancy is most frequently a sign of scurvy. Sugar is often present in the urine of infants without any special reason to account for it; it is probably derived from the lactose in the milk, especially when there is a greater consumption than can be assimilated.

The presence of *urates* and *uric acid* has been referred to.

It is usually indicative of a gouty diathesis, especially when the parents present such a history.

*Indican* is often found in the urine of children, probably a result of intestinal putrefaction (small intestine). Its frequent association with epilepsy is its most important feature. The majority of specimens of urine from artificially-fed infants that I have examined contained this substance in excess. Correctly speaking, it is an indoxyl-potassium-sulphate.

## CHAPTER III.

### THERAPEUTICS.

In the treatment of the sick a drug should never be given, unless specific indications for its use exist. Even under these conditions medicines should not be prescribed until every detail of hygiene and diet has been attended to. Moreover, if it is possible to obtain a therapeutic result by means of such simple non-medicinal measures as hydrotherapy, massage and exercise, it is not only superfluous but irrational to subject the system to drug effects. The physician who prescribes small doses cannot shield himself from this criticism by retorting that the drug will not injure the patient and, therefore, it will make no difference.

Rational therapeutics presupposes accuracy in diagnosis. Our drug pathogenesis, *i. e.*, the reliable symptoms of our *Materia Medica*, is based on the pathological conditions and physiological disturbances induced in the healthy human organism by the administration of the drug in sufficient quantity to induce these phenomena. Our method of prescribing is based on the rule that a drug capable of producing certain pathological effects, with the consequent appearance of certain symptoms arising therefrom, is capable of controlling and removing identical symptoms when encountered in a sick individual. Our dosage is based on the observation that while large doses aggravate these symptoms, smaller ones act curatively.

This mode of practice, however, like every other therapeutic system, has its limitations. Circumstances arise, as Hahnemann himself points out (*Organon of Medicine*, § 67), "where danger to life and imminent death allow no time for the action of a homœopathic remedy." It is largely a matter of opinion as to just what constitute the indications for physiological

interference. Errors are made on both sides. The early resort to powerful stimulants in all fevers and the free use of the depressing "antipyretics" has undoubtedly done more harm than the absolute neglect of taking the state of the heart and the height of the fever into consideration and relying exclusively upon the "indicated remedy."

"In order to obtain indications for treatment, make a diagnosis. The art is becoming both more accessible and, through honest and hard work, more easy with the aid of modern methods (JACOBI)." By "diagnosis" is not meant the mere tagging of a name to a disease—anæmia, jaundice, dropsy, even more exact nomenclature, such as lobar pneumonia and typhoid fever, is not a diagnosis. Recognize the patient's vital resistance, the state of his heart muscle. Will it see him through unaided? Is your remedy sustaining it or will you have to resort to more energetic means? Is it possible to keep up nutrition by the ordinary means? Can we foretell and prevent complications? This is diagnosis in the modern sense of the term, and when Gerhardt says "without diagnosis no intelligent therapy," he does not refer to the mere detection of physical signs of disease.

**Stimulants.**—In a previous chapter (page 18) it has been pointed out that cold water is a powerful stimulant under certain conditions. This method of stimulation is, however, not always available or applicable.

*Alcohol* is well borne by young children and is one of the most generally used stimulants we possess. Aside from its sustaining action upon the heart it is a food in the sense that it is oxidized in the body and thus spares tissue waste. Alcohol does not materially affect the blood pressure and is, therefore, not to be relied upon in a rapidly failing heart or in collapse. Its use is rather to ward off such an emergency than to meet it. The antidotal action of alcohol in the various toxæmias is one of its most valuable attributes; this applies especially to septic conditions and low typhoid states. In diseases of short duration, however, with high fever, it is sel-

dom indicated; in fact, it is useful only when such cases become adynamic. In gastro-intestinal affections and even in nephritis it is not contra-indicated providing it be cautiously administered and well diluted.

The indications for the use of alcohol in a continued fever are a soft, rapid pulse and a failing of the muscular element in the first sound of the heart. The appearance of restlessness and delirium, dry tongue, distended abdomen and pulmonary congestion calls for an increase in the dose. In diphtheria, alcohol may safely be used from the very beginning in moderate dosage, and increased as the necessity arises.

In a young infant, ten to twenty drops of brandy well diluted, may be given every two to three hours when urgently required. An infant one year old may take half a drachm every two hours; this can be increased to one drachm, if necessary. A child from three to five years old may take as high as two drachms every two hours in low typhoid conditions. When the odor of alcohol can be detected on the breath we may know that the patient is fully under its influence and repetition of the dose becomes unnecessary.

*Camphor*.—As a quick, diffusible stimulant there is nothing better than *Camphor* when urgent symptoms are to be met. The picture calling for *Camphor* is one of collapse. Drop doses of the tincture should be used, or what is better, camphorated oil injected subcutaneously. Personally, I prefer the neutral solution of *Camphor*. This is of the same strength as the oil, namely, 12 ½ per cent. In a young infant two to three minims suffice. A year-old babe may receive five minims, while a child from three to five years can safely be given ten to fifteen minims. If no result is seen within fifteen minutes, the injection may be repeated in a somewhat smaller dose.

*Digitalin, Strychnia*.—Cook (*American Jour. Med. Sciences*, April, 1903) has demonstrated by means of observations with the sphygmomanometer of Riva-Rocci that *Digitalin* will raise the blood pressure in cases of failing circulation within

fifteen minutes, and maintain it at a safe point for several hours. It is, therefore, to be preferred to *Strychnia* when prompt results are demanded. *Strychnia*, on the other hand, maintains the pressure longer and better than *Digitalin* and should be used to reinforce the latter when heart failure is to be averted.

Cook's experiments have also demonstrated that the blood pressure is a most valuable guide in showing us when stimulation is actually necessary. Some cases that appeared to require it were found to have almost normal pressure and consequently the stimulant was stopped, while others that did not betray their critical condition by the ordinary signs were found dangerously near the point at which life ceases. The dosage of these drugs is one four-hundredth grain hypodermically in a young infant, and one two-hundredth grain in a child.

**Prescribing.**—The method of prescribing for children resembles the method of diagnosing their ailments in that we are dependent entirely upon objective signs for reliable indications for a remedy. Far from being a disadvantage, this really gives us a better opportunity for practicing scientific therapeutics because the source of error resulting from the unreliability of "subjective sensations" is removed. Moreover, the data upon which we prescribe are based on pathological states which we interpret as "objective symptoms," and, therefore, more demonstrable and tangible than the other class of indications.

Prescribing is practically diagnosing the remedy, and we should go about it in much the same manner. The family history, the constitution and temperament, previous history, mode of onset, etc., all offer clues to the proper remedy. While the diathesis and temperament cannot be accepted as genuine indications for a remedy, still we know that certain individuals are especially susceptible to certain drugs, that the state of their nutrition calls for certain remedial agents, and that distinct moods and peculiar states of the mind and nervous system come within the sphere of drug action.

Each diathesis has a group of remedies wonderfully adapted to its needs; the temperaments are well defined in our *Materia Medica*, and the constitution likewise, whenever it presents special susceptibility to a drug. This has been noted under the clinical indications of our symptomatology.

The previous history often points to a constitutional remedy; thus, late appearance of the teeth and a late closure of the fontanel will suggest the need for *Calc. phos.*; the opposite condition will rather point to *Calc. carb.* Former skin eruptions, especially when combined with snuffles and sore mouth, will probably indicate one of the *Mercuries*; or if the child comes to us with a history of having been salivated, *Hepar*, *Nitric acid* and the *Iodide of potash* will suggest themselves. Certain remedies we know to be especially useful in removing the remote effects of various ailments; thus, *Sulphur* after pneumonia; *Arnica* when there has been a trauma; *Silicea* sometimes after vaccination and *Ignatia* after fright. Again, disturbances resulting from the abuse of such drugs as *Iron* and *Quinine* often require *Pulsatilla*; after anodynes, purgatives, cough mixtures and the like *Nuxvomica* will prove useful.

We should observe the position assumed by the child during sleep and waking. This often offers valuable suggestions for a remedy. For example, lying quietly upon the affected side is a characteristic indication for *Bryonia*.

The condition of the skin, whether dry or moist, hot or cold, red or cyanotic; also, if eruptions be present, their characteristic features—all are important to the prescriber.

The physiognomy may offer suggestions; the knitting of the brow pointing to headache, the fanlike motion of the *alæ nasi*, indicating dyspnœa.

The character of the cry may indicate *Apis*, when there is effusion into the brain, it may point to *Bell.*, *Acon.* or *Puls.* if otitis is diagnosed, or *Mercury* and *Kali bichromicum* when syphilis is suspected from the hoarse, feeble tone. Sudden hoarseness should, however, lead us to suspect the advent of



croup, when we naturally choose between *Aconite*, *Spongia* and *Hepar*.

In examining the chest we aim to define the character of the râles present, and are thereby able to differentiate remedies. Thus, *Ant. tart.* and *Ipecac.* are differentiated by the predominance of and finer character of the râles in the latter; in *Ant. tart.* there are coarse râles, in the larger bronchial tubes from the accumulation of mucus which the patient is unable to cough up. The discovery of consolidation, effusion and friction sounds will also aid us in prescribing.

Objective signs in cardiac disease are valuable aids in prescribing, only to mention *Acon.* and *Rhus tox* in hypertrophy; *Spigelia* and *Bryonia* in endocarditis; *Glonoin* for the high arterial tension and *Cactus* in valvular affections.

In prescribing for diseases of the nervous system we must carefully differentiate the various conditions occurring here. Thus, in differentiating cerebral anæmia and hyperæmia from inflammatory processes our prescribing will necessarily be more accurate and successful.

After we have decided that the meninges are involved, a number of well-known remedies will immediately present themselves. To differentiate between them we must take into consideration the degree of fever and cerebral congestion; the presence or absence of convulsions, photophobia and strabismus; the psychical state, manifested by the disposition, character of sleep and state of consciousness; delirium or coma. This, together with a general survey of the patient, gives the data for finding the similimum.

And so the special senses, the alimentary tract and the genito-urinary tract are all to be carefully studied in the manner above detailed, in order to gain the requisite knowledge for making a prescription. The results of such prescribing bring their due reward; it is time well spent in fruitful labor.

**Dosage.**—The dose, while an important question, is not the principle upon which Homœopathy is based. Our unfair critics would have it believed that Homœopathy and micro-

therapy are one and the same thing. The fundamental principle of Homœopathy, however, is the sound deduction formulated by Hahnemann as the general therapeutic rule of practice, *similia similibus curentur*, and the dose recommended was the smallest one that would act curatively without aggravating the condition for which it was prescribed. There is no necessity, therefore, for invading the realm of the infinitesimal in order to practice Homœopathy. In fact this method of dosage was adopted by Hahnemann himself only in his later years.

To the beginner, and especially to those not in sympathy with the theory of attenuation, small doses of the tincture and the lower dilutions are to be recommended. When employing insoluble substances, the lower triturations may be used. Let the dose just fall short of producing medicinal aggravation, and if the remedy be homœopathically indicated, a curative result will follow. Accordingly, the liquid remedies, excepting the very poisonous ones, may be administered in doses of one to two drops of the first or second decimal dilution, repeated every one to two hours in acute conditions, without fear of doing any harm. In young infants the second and third decimal dilutions are usually preferable. The same may be said of triturations; but it is reasonable to suppose that insoluble and apparently inert substances like *Silica* and the *Carbonate of Lime* are more active when their molecules are mechanically separated than in the crude state. The interesting and convincing experiments conducted by Dr. Percy Wilde, published in the *Journal of the British Homœopathic Medical Society*, January, 1902 ("Energy in its Relation to Drug Action"), prove conclusively that the process of trituration induces decided changes in the physical properties of the substance thus treated. There seems to be no doubt that this process converts apparently inert substances into a state in which they can enter into chemical combination with certain cells of the human economy for which they possess a selective affinity. If, therefore, we desire to obtain the thera-

peutic action of one of these remedies, we must give it in a finely subdivided state, such as the third to sixth decimal triturations represent. On the other hand, when we desire to obtain simply the nutritive or chemical effect, as in using *Iron* in anæmia, a much larger dose becomes necessary. The action of *Ferrum phosphoricum* in the third decimal trituration in acute bronchitis is essentially different from the action of *Ferrum reductum* crude or in the first decimal trituration in anæmia; in the former the action is medicinal, while in the latter it is chemical.

Triturations are usually dispensed in tablet form, each tablet representing one grain of the triturate. In acute conditions, a tablet may be administered every one to two hours; in chronic affections, two tablets four times daily is the usual dose. Naturally, such poisonous substances as *Bichloride of Mercury*, *Cyanide of Mercury* and *Arsenious acid* must be given with caution when used in the third decimal trituration.

## CHAPTER IV.

### INFANT FEEDING.

A comparison of the results obtained by artificial feeding and breast feeding indicates conclusively that, as ordinarily practiced, the artificial method fails to supplant successfully nature's method.

The question naturally arises, can a child be weaned with any degree of safety before the usual time, and can those who are deprived of breast milk from the very beginning of their existence be spared the gastro-intestinal derangements and the later constitutional manifestations of faulty nutrition which are almost universally the lot of hand-fed children?

A close study of the subject of infant feeding reveals the fact that nature can be imitated so closely by carefully and intelligently conducted methods that but very slight, if any, difference in results should occur. In the first place, we must study the chemical composition of human milk, and furnish the child with a substitute having a similar composition. Secondly, the food must be served perfectly sterile and of the temperature of breast milk, as the latter is entirely free from pathogenic and fermentative micro-organisms when secreted from a healthy breast, beside being of the body temperature. Thirdly, the proper quantity must be administered, and at regular and suitable intervals. If these conditions are carried out, artificial feeding is robbed of its terrors, and becomes a boon to infants and to sickly and delicate mothers who are not able to stand the drain of nursing.

### HUMAN MILK STUDIED IN COMPARISON WITH OTHER MILKS AND FEEDING MIXTURES.

Human milk is an alkaline fluid, bluish-white in color, of watery consistency and sweetish taste. It contains a slightly

lower percentage of total solids than cow's milk, and considerably less proteids, but a higher percentage of lactose (sugar of milk). This accounts for the difference in appearance and taste. The amount of fat is about equal in both, unless we take into consideration the milk of special breeds of cows, such as the Jersey, in which the fat may be as high as 5 per cent., more than 1 per cent. above average human milk.

The distinctive feature of human milk is its apparent low percentage of proteid, as compared with other milks. The amount of this nitrogenous element ranges between 1 and 2 per cent., the average obtained by Cautley from a large number of analyses being 1.93 per cent. Cow's milk contains almost uniformly 4 per cent. of proteid. In good dairy milk, where we obtain a mixed product from many cows, it seldom varies from this standard, and it can be kept so by the proper feeding and management of the herd. Mother's milk presents a much greater fluctuation, owing to the highly susceptible nervous system of the human subject.

There is, however, a difference in the proteid, aside from percentage, for the proteid of human as well as of cow's milk is not a single body, but can be resolved into caseinogen and lact-albumin, two bodies of totally different character and composition. In cow's milk the proportion of caseinogen to lact-albumin is four to one; in human milk, two to one.—(KÖENIG.)

The caseinogen of cow's milk is precipitated by acetic acid or by a saturated solution of magnesium sulphate. The lact-albumin is not affected by these reagents, but precipitates with tannic acid and by boiling. It is the chief constituent of the scum which forms on boiled milk. To estimate the percentage of lact-albumin in a given specimen of milk it is first necessary to precipitate the caseinogen with acetic acid, filter, and in the filtrate the lact-albumin can be estimated by tannic acid.

The caseinogen found in human milk forms a much finer curd than that of cow's milk when coagulated with the rennin

of the gastric juice. Again, the greater proportion of caseinogen to lact-albumin in cow's milk is another factor making it less digestible and less suitable for the infant.

Ass's milk more closely resembles human milk in the amount of proteids present, containing according to an analysis by Dujardin-Beaumetz, 1.23 per cent. proteids, 6.93 per cent. lactose, and 3.01 per cent. fat. The objection to its use is the difficulty of obtaining it and the low proportion of fat present.

The fat-globules of human milk are smaller than those of cow's milk, but aside from this there is no material difference in the cream of the two.

The reaction of human milk is alkaline, while cow's milk is usually acid by the time it reaches the consumer. This acidity is often a source of considerable disturbance in the child's digestion, but the difficulty is controllable, as it is a simple matter to recognize this condition of the milk and correct it.

The following table presents a comparison of human and cow's milk, constructed from the average of a large number of analyses by competent chemists.

*Standard Comparative Table of Human and Cow's Milk*  
(CAUTLEY, "The Feeding of Infants.")

	Cow's Milk.	Human Milk.
Water, . . . . .	87.	87.46
Solids, . . . . .	13.	12.54
Proteids, . . . . .	4.06	1.93
Fat, . . . . .	3.70	3.62
Lactose, . . . . .	4.48	6.75
Salts, . . . . .	0.76	0.26
Reaction, . . . . .	Acid.	Alkaline.

A great variation is found in the results obtained by different observers in analyses of human milk, the fluctuations in the percentages of proteids and fat being very marked at times, even in the same subject. Rotch cites a case in which the proteids rose from 2.53 to 4.61 per cent. in a wet-nurse, from being fed on a richer diet than she had been accustomed

to. Again, one observer will report having found 2 per cent. of proteids, while another finds 1 per cent. In a series of analyses made by A. V. Meigs the proteids varied from .73 to 1.27 per cent.; fat from 2.4 per cent. to 9 per cent. In a series of careful analyses recently reported by Hofmann, of Leipzig, the percentages stand as follows: Proteids, 1.03 per cent.; fat, 4.07 per cent.; lactose, 7.03 per cent.; salts, 0.21 per cent.

This only demonstrates the fact that the human subject is a very sensitive organism, easily influenced by emotional factors, character of diet, amount of exercise, and certain physiological states, such as the recurrence of the catamenia or pregnancy.

#### MILK ANALYSIS.

When an infant fails to digest breast-milk, or does not thrive on it, before condemning the child's digestive functions, we should examine the milk. Under all conditions, when the food disagrees it becomes imperative to institute a chemical analysis and microscopical examination of the milk. The information sought need be no more than an estimation of the fat and proteid percentages, while the microscope reveals the number and condition of the fat globules, whether perfectly or imperfectly emulsified, showing also abnormal elements when present, *i. e.*, colostrum corpuscles in excess, pus corpuscles, micro-organisms. The physician is to be encouraged in making these examinations, and I can only repeat here what I have elsewhere pointed out (*The Dietetics of Childhood in Health and Disease, Trans. Amer. Institute of Hom.*, 1901, p. 398), that a milk analysis is by no means so complicated a procedure as is generally supposed, being in no wise more troublesome than an ordinary examination of urine.

First, we must obtain a sufficient quantity to judge of the *appearance* of the milk. If the quantity secreted by the breasts be insufficient for the infant's needs, the case is hope-

less from the beginning, unless we can increase it with galactogogues.

The *specific gravity* ranges from 1025 to 1035, the average being 1030. The *reaction* should be alkaline. The method of obtaining these data is identical with that employed in urinalysis, excepting that a smaller instrument for obtaining the specific gravity is preferable—a *lactometer*—(Fig. 23)

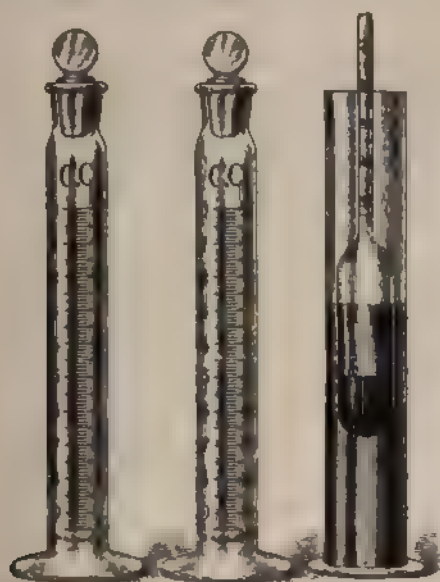


FIG. 23 \* -HOLT'S APPARATUS FOR EXAMINING WOMAN'S MILK CONSISTING OF A LACTOMETER AND CREAM GAUGES.

owing to the smaller quantity of specimen one is obliged to work with. The specimen is best obtained with a *breast-pump*, the middle portion of the milking being taken as an average sample.

The percentage of *fat* is the most vital point in question in passing judgment upon a sample of milk. The *cremometer* or *cream-gauge*—(Fig. 23) an instrument in which the milk

\*Made by Eimer & Amend, New York City



is allowed to stand until the cream rises to the top, when the percentage can be read off—offers a simple but not very accurate method of obtaining the fat per cent. Five per cent. of cream is equivalent to 3 per cent. fat by this method (HOLT). The separation of the cream, however, does not depend alone upon the amount of fat present, but is influenced by such physical states as the temperature, size of fat globules, and specific gravity of the milk (CONRAD), making it uncertain and unreliable.

The *lactobutyrometer* of Marchand gives more accurate results. The *milk-tubes*, supplied with the centrifuge, are constructed on a principle similar to that of Marchand's instrument, and offer a rapid and convenient method for obtaining the fat percentage. The lactobutyrometer is a graduated tube into which five c.cm. of milk are poured, together with a few drops of *liquor sodæ*, after which five c.cm. of *sulphuric ether* are added, and the fluids thoroughly intermixed by gentle agitation. *Alcohol* (90 per cent.) is then added in the same quantity—marks upon the tube indicating the proper amount of each element used—the tube is stoppered, again shaken, and placed in warm water for half an hour. The fat separates into a distinct oily layer that floats to the top, where the percentage can be read off by the scale on the tube.

The estimation of the amount of *proteids* is of less importance, and the data furnished by the character of the child's stools are usually sufficient. An approximate estimate of the percentage of proteids is obtained by comparing the *specific gravity* of the milk with the *fat percentage*. The specific gravity is elevated by proteids and lowered by fat. If, therefore, fat be deficient and the specific gravity low, we must infer that the amount of proteids must also be deficient. A high specific gravity with normal fat would, on the other hand, indicate an excess of proteids.

A *microscopical examination* should reveal a preponderance of small, uniformly-sized fat globules, indicating thorough

emulsification; they should be present numerously in the microscopic field. After the third week the milk must be free from all cell-elements, of which the colostrum corpuscle is an example, being the remnant of protoplasmic bodies originating from the cells of the mammary gland acini.

**Cow's Milk.**—A few simple facts applicable to *cow's milk* are worthy of mention, as it often becomes necessary to decide whether a given sample of milk is a suitable food for the infant. In the first place, a quart of milk standing for six hours after milking in an ordinary milk bottle should show a layer of cream in the neck of the bottle six inches deep. This cream contains on an average 12 per cent. of fat, but it varies in richness in the different layers; the top ounce may contain 25 per cent., and the sixth ounce only 5 per cent. fat (Fig. 25). (CHAPIN.) In any quart bottle of milk on which cream has risen the *top nine ounces* will contain about three times as much fat as the whole milk contained, and the *top fourteen or fifteen ounces* about twice as much (CHAPIN). This fact is taken advantage of in the top-milk method of home-modification of milk, known as "Chapin's method," and to my mind, the most practical as well as accurate method at our command.

**Impurities.**—Pathogenic bacteria gain entrance into the milk either with dirt acquired during milking, *i. e.*, stable filth, or direct from the milker, or through the use of impure water in washing out the containers. The germs that cause the milk to turn sour come mostly from the first few jets from the cow's teats. If these first jets are rejected, perfect cleanliness observed, and the milk cooled below 60° F. immediately after milking, it is practically sterile, will keep satisfactorily and can be fed without sterilizing excepting in hot weather.

**Preservatives.**—The presence of a preservative, *e. g.*, formaldehyde, should be suspected in a milk which does not curdle within twenty-four hours when placed in a stoppered bottle and kept in a warm place.

**Reaction.**—The quantity of lactic acid that has formed in

the milk by the time it reaches the consumer is a good index of the amount of care that has been exercised in handling it. The sense of smell and taste is hardly accurate enough to afford a reliable test in determining the quality of the milk, and the simplest and most practical means of deciding the fitness of a specimen of milk is offered by the "Ideal Milk Testers." One of these tablets is dissolved in an ounce of water, and the resulting pink solution is added, a teaspoonful at a time, to a teaspoonful of milk until the mixture becomes permanently decolorized. By following the scale accompanying these testers we can draw our conclusions. The reaction depends upon the neutralization of the lactic acid in the milk with Sodium bicarbonate, the indicator being phenolphthalein.

#### CAUSES INFLUENCING THE COMPOSITION OF BREAST MILK.

The milk obtained at the beginning of a milking is known as the *fore-milk*; it is watery and poor in fat. Next comes the *middle-milk*, and lastly the *strippings*. The middle-milk should be used for an analysis when the contents of the entire udder or breast cannot be obtained. The strippings are especially rich in fat, and also contain a higher percentage of proteid than the fore-milk.

The intervals at which the breast is emptied markedly influences the composition of the milk. The longer the interval, the more watery the milk, and the more frequently the breast is used, the more concentrated the milk becomes. When the bad habit of putting the child to the breast every one or one and a half hours is persisted in, a veritable "condensed milk" will eventually be secreted, which, it is needless to state, cannot be digested by the infant. It may be laid down as a maxim that the more frequently the child is nursed, the more indigestible the milk becomes. The over-stimulation of the mammary gland leads to an increased secretion of proteids, while the percentage of fat is also augmented, the milk resembling the *strippings* in this respect.

Food and exercise exert a marked influence upon the composition of milk. The richness of the milk, that is the amount of fat, is increased by a nitrogenous diet, and is decreased by an excess of fatty foods, owing to the diminished metabolic activity induced by such a diet.

The proteids are increased, together with the fat, on a liberal proteid diet; also from increased frequency of nursing, as has been pointed out, and especially when a liberal diet is enjoyed, together with insufficient exercise. This is frequently a source of much trouble with wet-nurses, who, entering upon their new duties with privileges not formerly enjoyed, a diet and sedentary occupation to which they are not accustomed, soon secrete a milk hardly to be distinguished from rich cow's milk in its chemical composition and indigestible character. To correct this condition the nitrogenous food must be considerably cut down and sufficient exercise taken until the percentages become normal.

The effect of alcohol moderately used is not injurious to the milk, and in some instances is highly beneficial to the mother. Some of the malt liquors certainly act as galactogogues, and the amount of fat is slightly increased by the use of alcohol. When used in excess, serious gastro-intestinal disturbances in the infant may arise.

Menstruation sometimes induces changes in the milk which cause it to disagree. Rotch reports a case in which the proteids rose to 2.12 per cent., while the fat fell to 2.02 per cent., rendering the milk difficult of digestion and interfering with the regular rate of progress in the child's weight. On the other hand, Schlichter, who made analyses in thirty-three cases of menstruating women, concludes that diarrhoea and colic should rather be looked upon as coincidences, for he found no decided alterations in the milk.

Should the mother become pregnant it is not advisable to continue breast feeding, as the drain upon her system is usually too great to be borne by the average woman, and, besides, there is danger of inducing miscarriage. Moreover, the child

usually ceases its progressive gain in weight, and evinces signs of not being satisfied with its nourishment. If it is necessary, however, to temporize on account of the delicate state of the child and the time of year, it may be suckled to the sixth month, and then partial weaning instituted. This will rarely be necessary, for with our present knowledge of infant feeding, and the accurate and safe methods at our disposal, the dangers of weaning, formerly so much feared, can be reduced to a minimum.

The *wet nurse* is not ordinarily a desirable substitute for the mother's breast, nor is it always possible to obtain one that will conform to the requirements necessary in fulfilling such a charge. In the first place, a careful medical inspection must be instituted in order to be certain that no evidences of constitutional or contagious disease are present. Secondly, there must be a sufficient quantity of milk secreted and the breast and nipples must be in a normal condition. It is also important that the stage of lactation shall correspond closely to the age of the infant to be nursed; especially disadvantageous is it for an older infant to suckle from a nurse in the early period of her lactation, the converse condition being less unfavorable. (Baginsky, *Lehrbuch der Kinderkrankheiten*.) The prominent influence of diet and exercise upon the composition and digestibility of the milk has been referred to above and the strictest regulations must be enforced in this direction. Highly seasoned food is also to be avoided, as well as all acid fruits and salads, indigestible vegetables, and the free use of alcoholics.

#### THE MODIFICATION OF COW'S MILK.

It has been pointed out that cow's milk in its raw state is not a suitable infant food for two reasons, namely, on account of the excessive amount of proteids and their indigestible character as compared with those found in mother's milk, and the contamination by micro-organisms so universally present. To overcome the first objection, we must put the

milk through a process of modification, in which the percentages of its proximate principles are made to conform to the standard composition of human milk. Sometimes, however, it will be found necessary either to reduce or increase the percentage of these elements, the necessity and indication for which will be discussed later.

By referring to the table shown below an idea of the difference between human and cow's milk may be obtained. Regarding the percentage of proteids in human milk, no fast rule can be laid down, and it has been shown how analyses by different chemists vary, and how strongly diet, exercise and constitutional disturbances influence the composition of the mammary secretion.

The following table represents a fair working basis for this problem :

	Human Per cent	Cow Per cent
Proteids, . . . . .	12	4
Fat, . . . . .	4	4
Sugar, . . . . .	7	4.5
Water, . . . . .	87	86
Reaction, . . . . .	Alkaline.	Acid.

Gaertner (*Therapeutische Wochenschrift*, May, 1895), has devised a practical method of modifying cow's milk to approximate human milk in composition by the use of the centrifuge. Equal parts of distilled water and milk are put into the centrifuge and separated into two portions, one containing all the cream, beside 2 per cent. of casein. A tablespoonful of sugar of milk is added to each half litre of this "Fettmilch," which renders it very similar in composition to human milk and a very useful food for most infants. Fischer (*Medical Record*, Dec. 11, 1897) has recently reported a series of cases, among them entero-colitis, gastro-enteric catarrh and athrepsia, which improved rapidly under a change of diet to this formula. A preparation very similar to this can be made at home simply by allowing the milk to sepa-

rate by standing, this method having long been in use, and already warmly recommended by Guernsey (*Obstetrics*, p. 622; Phila., 1867).

It is, however, often not only desirable, but absolutely necessary to vary the percentages of the proximate principles, or to imitate closely a given formula, in which case we must have a definite mode of procedure, which is at the same time simple and practical in its application. A method of modifying cow's milk to conform with the indications of each case, which I have used with signal success both in private practice and in my hospital work, has been reported on a former occasion, under the title *The Artificial Feeding of Infants with Synthetical Milk* (*Hahnemannian Monthly*, Feb. 1898). It is, however, too complicated for use in private practice, and the methods of obtaining varying percentages of fat and proteids detailed below will answer for all ordinary purposes.

A word as to the use of *milk sugar* and *cane sugar*. It is held that lactose, being the natural sugar found in milk, and being more assimilable and less liable to undergo fermentation than cane sugar, should always be used in artificial foods. The objections to cane sugar are, however, rather theoretical than practical, and we know from the condensed-milk baby that cane sugar has great fattening properties. Besides, it does not readily undergo fermentation, its use as a preservative demonstrating this fact. Again, lactose, on account of its property of being converted into lactic acid, which again may be converted into butyric acid, is often objectionable. It may also act as a laxative. Jacobi (*Archives of Pediatrics*, Oct., 1901) opposes the use of milk sugar on these grounds and also because it is difficult to obtain a pure article. He doubts the identity of the sugar of milk from the cow with that of human milk and calls attention to the danger resulting from the presence of lactic acid in the alimentary tract. This acid throws out of solution the casein of the milk and causes diarrhœa. It also increases the elimination of



lime-salts from the tissues by the kidneys and lays the foundation for the development of rickets and malnutrition.

In many of the larger cities milk laboratories have been established, where the physician may have made up and served a formula of any proportion of fat, proteids and sugar that he wishes to prescribe. These laboratories are a great convenience, and they are conducted on the lines laid down by Rotch, who was the first to advocate mathematically accurate percentage feeding. But the results of prolonged feeding with laboratory milk are as a rule unsatisfactory. Proteid digestion is defective and the fat does not seem to be assimilated. Evidences of mal-assimilation and malnutrition are commonly observed and I have seen rickets develop under these circumstances. Starr (*Diseases of the Digestive Organs in Children*) speaks with disapproval of the use of laboratory milk. He has observed the development of gastro-intestinal catarrh in many instances and even of scurvy. In his opinion it is the complete separation of the fat and proteids in the preparation of the milk formulæ that interferes with the emulsification and digestibility of the fat. Holt, on the contrary, does not believe that this is of any practical importance and speaks favorably of laboratory milk.

The theory upon which this mode of feeding is based is correct, but personally I prefer the home modification of milk. There is no doubt that the results obtained from feeding a properly diluted top-milk—whose fat percentage can be easily gauged—are far superior to those obtained from the use of a modified milk consisting of a readmixture of the milk elements after mechanical separation, that have again separated because the food must be prepared in the laboratory anywhere from twelve to twenty-four hours before the last bottle is fed to the child, and that must be rigorously sterilized, or be sour and unfit for use by the time the child is ready to take it. It is very likely that the success of laboratory milk has depended more upon the purity of the article than upon the strict adjustment of percentages.



The following rule for diluting cow's milk expresses the underlying principle of the home modification of milk: For an infant *under two weeks* it should be diluted five times; from *two weeks to six weeks*, four times; from *six weeks to three months*, three times; from *three months to four months*, twice; and from *four months to nine months*, once.

If, however, we were to use ordinary milk, the result would be a deficiency of fat and lactose in the food, for which reason a milk containing 10 per cent. fat (a *10 per cent. "top-milk"*) must be employed up to the third or fourth



FIG. 24.—DIAGRAM SHOWING THE PERCENTAGE OF FAT IN WHOLE MILK AND IN THE UPPER LAYERS OF SET MILK USED IN MODIFYING MILK. (AFTER HOLT.)

month and a *7 per cent. top-milk* from the fourth to the ninth month. After that, ordinary milk, slightly diluted, may be administered (Fig. 24).

The water used as a *diluent* must contain *milk-sugar* in the proportion of one ounce to every twenty ounces of food and about the same proportion of lime-water to neutralize the acidity of the milk. In the later period of infancy *granulated sugar* may be used instead of milk sugar. About one-third less than the amount of milk sugar specified should be added. It is always best to boil the water for ten minutes

and dissolve the sugar therein while still hot. Lime-water, however, is decomposed by high temperature.

*Ten per cent.* milk is obtained by dipping off the upper ten ounces from a quart bottle of milk that has stood on ice for from four to six hours (until all the fat has risen to the top)

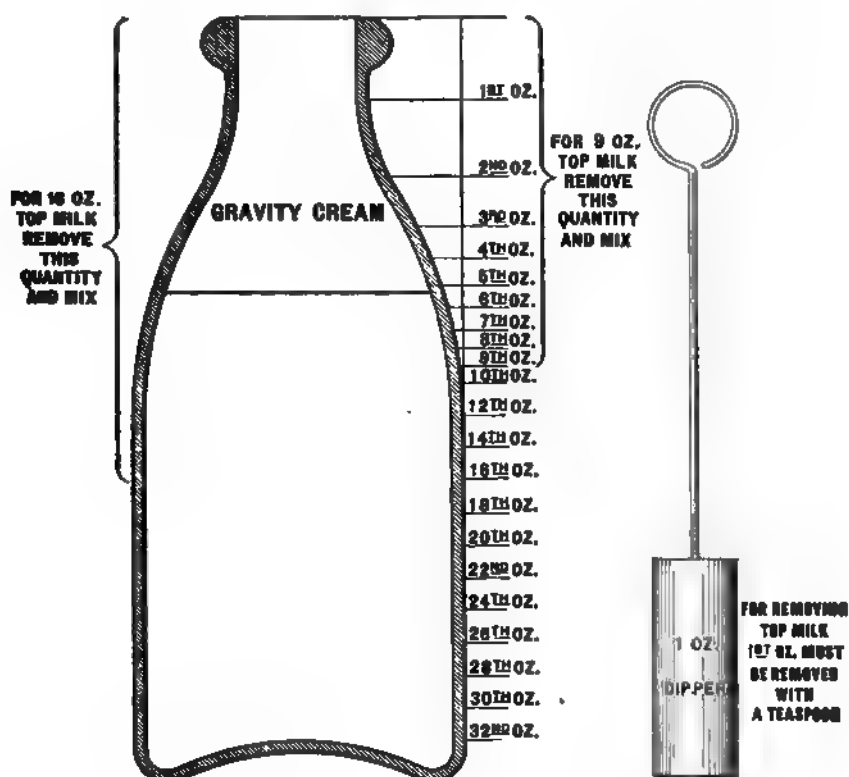


FIG. 25.—DIAGRAM SHOWING FAT PERCENTAGE OF DIFFERENT LAYERS OF SET MILK AND CHAPIN'S MILK DIPPER. (CHAPIN, THEORY AND PRACTICE OF INFANT FEEDING.)

with a Chapin milk dipper. (Fig. 25.) Carefully pouring off this upper layer into a glass graduate will answer when the dipper cannot be obtained but the results are by no means as accurate. The formula of this upper third is approximately: fat, 10 per cent.; proteids,  $3\frac{1}{2}$  per cent.; the ratio

of fat to proteids being three to one. When not obtainable in this manner the nurse may be instructed to take *equal parts of plain* (whole) *milk* and *ordinary cream*, which contains 16 per cent. fat. The resulting mixture is a 10 per cent. milk.

*Seven per cent. top-milk* is obtained by dipping off the upper half (16 oz.) from a quart bottle of set milk. Its equivalent in cream and milk mixtures is *three parts whole milk and one part ordinary cream*. The formula of this mixture is 7 per cent. fat and  $3\frac{1}{2}$  per cent. proteids, the ratio of fat to proteid being 2 : 1. The composition of the individual layers of "creamed" or "set-milk" are shown in Fig. 25.

It is advisable to make up the twenty-four hours' quantity in the morning, pasteurize it, and then keep the bottles on ice (they must be kept below 40° F.). When, however, fresh milk can be obtained twice daily there may be an advantage in making up half the daily amount at a time. This is especially the case when raw milk of undoubted purity is being fed.

When the child experiences difficulty in digesting the casein of the milk we should use barley-water as a diluent. As the young infant has but feeble starch-digesting power it is advisable to predigest the barley solution with one of the commercial diastatic ferments, such as Cereo, Forbes' Diastase or Maltine. A teaspoonful of diastase is added to a quart of barley water (two heaping tablespoonfuls barley flour, one quart water; boil fifteen minutes and strain) when the same has cooled sufficiently to be tasted.

Personally I prefer the top-milk method to the use of milk and cream mixtures, as it is cleaner and simpler. The table given below indicates the times of dilution for the different ages, together with the kind of top-milk to be used, the time for feeding and the amounts to be fed. By *times of dilution* is meant *how many times as much water as milk* is to be used. Thus, 5x dilution means one part top-milk and five parts water. When a *milk and cream mixture* is used the sum of the constituents of the food is equal to the amount of top-

milk used for a similar formula. Thus, in making up a formula to contain four ounces 10 per cent. top-milk and sixteen ounces water, if milk and cream were to be used it would require two ounces of each.

The composition of a given formula is readily calculated by simple division. For example, if we dilute 10 per cent. top-milk (containing 10 per cent. fat and 3½ per cent. proteids) two times, the top-milk will represent one-third of the mixture. The mixture therefore contains 3 ⅓ per cent. fat and 1 per cent. proteids, approximately.

In the early months of infancy the proper ratio of fat to proteids under normal conditions is 3 : 1. In later infancy the babe can digest more proteids and the ratio is changed to 2 : 1 with advantage.

Age.	Top Milk.	Times Dilutions.	No. of Feedings in 24 hours.		Intervals Between Feedings.	Quantity for One Feed.	Quantity for 24 hours.	Night Feeding 10 P. M. to 7 A. M.	Day Feeding.
1-2 weeks,	10 % fat	5x	10	2	hrs.	1-2 oz.	10-20 oz.	2	First Feeding, 7 A. M.
1½ months,	10 % fat	4x	10	2	hrs.	2-3 oz.	20-30 oz.	2	
2 months,	10 % fat	3x	8	2½	hrs.	3-4 oz.	24-32 oz.	1	Last Feeding, 10 P. M.
3-4 months,	10 % fat	2x	7	3	hrs.	4-6 oz.	28-42 oz.	1	
5 months,	7 % fat	1x	6	3	hrs.	7 oz.	42 oz.	0	
6-9 months,	7 % fat	1x	6	3	hrs.	8 oz.	48 oz.	0	

	3d to 14th day. No. 1.	2d to 6th week. No. 2.	6th to 12th week. No. 3.	3 to 5 months. No. 4.	5 to 10 months. No. 5.
Milk, . . . . .	1½	3	4	12	18
Cream, . . . . .	1½	3	4	5	6
Lime-water, . . . . .	1½	1½	1½	2	2
Water, . . . . .	15½	22½	22½	23	22
Milk-sugar, . . . . .	2½	3½	4	5½	6½
(Even tablespoonfuls.)	(20 oz.)	(30 oz.)	(32 oz.)	(42 oz.)	(48 oz.)

*Remarks.*—These quantities and percentages are only approximate, but they offer a standard by which the physician can be safely guided. If the child vomits shortly after finishing its bottle, it is either getting its food too rapidly or in too large quantities. *Regurgitation* of food between

In the table giving the amount of milk, cream and other ingredients it will be observed that the amount of milk and cream together represent the amount of top-milk that would be required to make up these formulæ. Thus, substituting top-milk for milk and cream, formula No. 1 calls for three ounces 10 per cent. top-milk; No. 2, six ounces 10 per cent. top-milk; No. 3, eight ounces 10 per cent. top-milk; No. 4, seventeen ounces 7 per cent. top-milk; No. 5, twenty-four ounces 7 per cent. top-milk.

In these formulæ the ratio of fat to proteids is retained as three to one up to the fourth month, the first containing 1.5 per cent. fat, 0.5 per cent. proteids, and 5.5 per cent. lactose. The second contains 2 per cent. fat, 0.7 per cent. proteids, 5.5 per cent. lactose. No. 3 contains 2.5 per cent. fat, 0.8 per cent. proteids, 6 per cent. lactose.

In Nos. 4 and 5 the ratio between fat and proteids stands two to one. No. 4 contains about 3 per cent. fat, 1.5 per cent. proteids and 7 per cent. sugar. No. 5 represents 3.5 per cent. fat, 1.75 per cent. proteids and 7 per cent. sugar (HOLT).

#### OTHER FOODS THAN MILK; WEANING.

During the *first year* a child may take *farinaceous food* in the form of thick, strained gruels prepared from barley, rice or oat-meal, and added in quantities of one to three teaspoonfuls to a bottle of milk. During the first half of infancy all farinaceous foods should be dextrinized, as the salivary glands and pancreas are not sufficiently active at this period of life to dispose of the starch. A thin gruel, such as *barley-water*, possesses, besides its nutritive value, the physical property of rendering casein more digestible, as has been already pointed out.

---

feedings, usually of sour milk, indicates excess of cream. *Constipation*, as a rule, indicates deficient cream or deficient quantity of food. *Curds* in the stool indicate excess of proteids or deficient proteid digestion, and calls for further dilution of the milk. *Colic* is a result either of proteid indigestion or too rapid nursing. A large, robust child naturally requires more food than a delicate, undersized child, and *vice versa*. Constant *crying* between feedings, when not due to pain, signifies hunger; this, together with insufficient weekly *gain in weight*, suggests an increased quantity or less dilution of the food.

*Beef-juice* is a valuable food for infants who are anæmic or who do not thrive well on milk alone ; also in scurvy. A teaspoonful may be given three times daily in the latter part of the first year ; earlier, half that amount, excepting in cases of scurvy, where more is necessary.

*Orange-juice* possesses decided antiscorbutic properties and should be administered regularly, a half to one teaspoonful three times daily, one hour before feeding, to infants taking sterilized milk or a proprietary food, or in cases of constipation. *Fresh grape-juice* is likewise beneficial.

During the first half of the *second year* five meals a day may be continued at intervals of four hours, the fifth meal being a bottle of milk at 10 P. M. Milk may be given in the bottle until the child is sixteen months old, when it should be taught to drink from a cup. Ten ounces of milk with a cereal (eight ounces of milk, two ounces of thick oatmeal or barley-water) will furnish the main food ; this can be given four times daily. At the noon meal a poached egg or some chopped broiled meat (rare) on alternate days should be added to the dietary. Stale bread and zweibach softened with milk are allowable, also fruit-juices, the soft portion of a baked apple and of stewed prunes. By the end of the second year, when all the teeth have made their appearance, a child will be able to take table-food of a light and digestible nature. Meat should only be allowed sparingly, however, and tea or coffee prohibited. The child should be encouraged to drink water freely.

*Weaning* should be begun at the end of the ninth month, providing the mother's condition does not demand that the infant be taken from the breast earlier. During the summer months it is often advisable to carry the child along a little longer to forego the dangers of summer-complaint. When, however, gradually done and the food carefully prepared there is no great danger in weaning. For the first few days a bottle can be substituted for a nursing ; as the child becomes accustomed to the bottle another can be added until

the breast is eventually dispensed with entirely. As a rule, it can be said that a babe just weaned from the breast will not be able to digest a mixture of cow's milk which a babe of the same age that was fed by hand from birth can digest. We must, therefore, begin on a somewhat weaker mixture than one recommended for the average case. By the sixteenth month the child should be weaned from the bottle and taught to drink from a cup, excepting the 10 P. M. feeding, which can be given it from a bottle in bed.

The following dietary is appended as a resume of feeding in later infancy:

*Diet from nine to twelve months:* whole milk, six ounces; barley-water, three ounces; granulated sugar, one drachm; a bottle every four hours (five feedings in twenty-four hours). Orange juice and meat juice also to be given as directed above.

*Diet from twelve to sixteen months:* whole milk, eight ounces; thick barley or oatmeal-water (gruel), two ounces; sugar, one and one-half drachms; every four hours (five feedings in twenty-four hours). At this period one semi-solid meal (a soft-boiled egg, cereal, milk pudding, zweibach soaked in milk) may be given once daily as a substitute for a bottle of milk; also a bottle of broth.

*Diet from sixteen to twenty-four months:* 7 A. M., one-half ounce orange juice; 7:30 A. M., a cereal, soft-boiled egg occasionally, eight ounces plain milk, bread and butter; 11 A. M., cup of broth with rice or barley, strained (if broth is to be given for dinner, this meal should be a glass of milk); 2 P. M., finely-cut meat every other day, cup of broth or glass of milk, baked potato, or boiled rice, or a well-cooked fresh vegetable, bread and butter, dessert (milk puddings, junket, custard, gelatin, stewed fruit); 6 P. M., cereal or bread and milk, stewed fruit.

During the *third year* the same schedule is observed but the variety and quantity of food is gradually increased, as all the teeth are present at this time the food can be of a more solid character and more meat allowed.

## THE INDICATIONS FOR VARYING THE PERCENTAGES OF THE PROXIMATE PRINCIPLES OF THE INFANT'S FOOD.

The character of the stool and the rate of progress in the child's weight are the data by which we must be guided in regulating the composition of the diet. Exceptionally breast milk disagrees with the child, the commonest source of disturbance being the increase in the proteids, as shown in Rotch's case, cited above. The symptoms pointing to this condition are vomiting of large curds, colic, constipation, or greenish stools containing tough curds. If the child persistently indulges in a food too rich in proteids, uric acid disturbances may develop. On the other hand, a deficiency of nitrogenous food leads to anæmia, a general laxity of the muscular system and checked physical development.

When fats are in excess, vomiting and diarrhœa may likewise be induced and the stools will contain fat in considerable quantity. But when normal amounts of fat are not disposed of we should suspect hepatic and duodenal disturbances or deficient pancreatic secretion excepting in the case of transient attacks of simple indigestion.

A deficiency of fat is very pernicious in its result, laying the foundation for the development of rickets and tuberculosis.

The chemical instability of the carbohydrates, of which group lactose, cane sugar and starch constitute the most important members, renders them especially liable to induce trouble, particularly when micro-organisms contaminate the diet. Through their fermentation lactic acid in the one case and alcohol, acetic, and butyric acid in another are formed in the alimentary tract, and the troublesome summer diarrhœas are largely traceable to this source. Children fed over a long period on foods rich in carbohydrates and poor in fat and proteids become large, flabby, and usually rachitic. They are anæmic and resist acute illnesses poorly—in fact, their plump bodies melt down to mere bony framework, almost as



a dropsy might rapidly disappear and leave an emaciated form behind.

The percentages of proteids and fat can often be modified in breast milk by regulating the mother's diet and habits. Still more readily they can be changed under artificial feeding, when the condition requiring such a change presents itself. The amount of lactose cannot be controlled in human milk. Whenever a breast-fed child shows signs of disordered digestion and impaired nutrition it becomes necessary to examine the milk, in order that the proper correction of the condition can intelligently be made.

#### THE INTERVALS FOR FEEDING AND THE QUANTITY REQUIRED BY THE CHILD AT DIFFERENT AGES.

The new-born infant is put to the breast as soon as the condition of the mother permits. Milk is not secreted before the third day, but *Colostrum*, which is a fluid rich in cells from the acini of the glands undergoing fatty metamorphosis, is present in sufficient quantity to appease the child's craving, and by its gentle laxative property empty the intestinal tract of the meconium. The child may be put to the breast every two hours during the first month, and if it be weakly, or show signs of not gaining progressively in weight, it may be nursed once during the night. Beginning at 5 A. M., and ending at 11 P. M., the child will have received ten nursings in all. During the second month the interval should be extended to two and one-half hours, and again one at night, if necessary. This will make eight nursings from 5 A. M. to 11 P. M. From the third month to the time of weaning, which should only under rare conditions be extended beyond the ninth month, the intervals will be every three hours, thus making seven nursings during the regular time.

These rules should be deviated from under no circumstances so long as the child is not seriously ill, and it is better to let it cry than give the breast before the prescribed time,

and awaken it when the time for nursing is due, until the child forms the habit of nursing regularly.

Some authorities recommend even longer intervals than those given above, but it will generally be found that the baby does satisfactorily under this *régime*, and where an individual case is found in which a more frequent or a more extended period seems necessary, it certainly will prove the wisest plan to make a change. The regularity of the feeding is the most important element. The advantages of this method over irregular feeding, or the little and often method, are too manifest to merit discussion.

When the infant is to be raised by hand from the beginning, it is well to commence with a 5 per cent. solution of milk sugar, sterilized, one ounce every two hours for the first day, until the bowels have been completely emptied and the child is taking the liquid well. It can then be put on a mixture containing about 0.8 per cent. proteids, 2 per cent. fat and 6 per cent. lactose; this is gradually increased as the child's digestion becomes stronger, until it is taking a formula corresponding to mother's milk.

In regard to the quantity to be given at a feed no fast rule can be laid down, for stomachs vary in size in children of the same age and weight, and a child of five months may have the feeding capacity of another at seven months. It has been estimated that the stomach capacity is equal to one-hundreth of the child's weight; Frowlowsky giving the following measurements:

One week, 1 ounce; four weeks, 2 ½ ounces; eight weeks, 3 1-5 ounces; twelve weeks, 3 1-3 ounces; sixteen weeks, 3 4-7 ounces; twenty weeks, 3 3-5 ounces.

The capacity of a hand-fed babe is, however, usually greater than the above, and the increase after the first month is more rapid than this table would indicate. The average quantity of food required by the infant at the different periods from birth to the time of weaning is given in the table on p. 93.

## THE STERILIZATION OF THE FOOD.

The first milk secreted from the human breast may contain a few varieties of staphylococci, as demonstrated by Cohn and Neumann, these micro-organisms having gained access to the milk-ducts through the nipple. After the breast has been thoroughly drained, the micro-organisms are flushed out of the ducts, and the milk is then usually quite sterile. Where, however, the breast is diseased, there may be an abundance of bacteria constantly present in the milk. This is the case in mastitis, especially in the parenchymatous variety, and in tuberculosis affecting the mammary gland. Under such conditions it is absolutely imperative to institute weaning.

Cow's milk is practically never sterile; indeed, not only diarrhoeal diseases are brought on by the use of contaminated milk, but epidemics of cholera, scarlet fever, typhoid fever and diphtheria, beside infection with tuberculosis and foot and mouth diseases, have been traced directly to the milk supply.

Of this group of infectious diseases cholera infantum and gastro-enteritis, respectively designated acute and subacute milk infection by Vaughn, and tuberculosis, are mainly to be feared, as they are constantly traceable to the method of feeding.

Regarding the last-named disease, it was formerly taught that tuberculous cows yield milk containing tubercle bacilli, whether or not the udder was affected; but the recent researches of Lewis Woodhead and Sidney Martin demonstrate that only milk from a cow with tuberculous udders is infectious.

Woodhead proved also that the temperature usually recognized as capable of destroying the tubercle bacillus, namely,  $75^{\circ}$  C, for a period of ten minutes, was not sufficient to render this micro-organism innocuous; and even when exposed twice that length of time, tuberculous milk produced in pigs a modified form of tuberculosis, manifesting itself as

chronic tuberculous glandular and joint affections, analogous to scrofulosis.

It is, therefore, highly important that all micro-organisms should be destroyed. Thoroughly boiling the milk for ten minutes renders it sterile from the clinical standpoint, or by subjecting it to a heat of  $212^{\circ}$  F. for an hour and a half in a steam sterilizer it will keep for several days in hot weather if carefully sealed. This process, however, affects the milk in its taste, physical properties and nutritive value. It tastes like boiled milk and is less digestible, the casein being rendered less coagulable by rennet and less soluble to the action of pepsin and pancreatin. Besides, it is believed that the prolonged use of sterilized milk invites the development of rickets and scurvy. According to Rundlett the albuminates of iron, phosphorus and fluorin are chemically changed by heating, the globulin or proteid molecule splitting away from the inorganic molecule, thus rendering these salts unassimilable (Fischer). For this reason many pædiatrists use raw milk—such known as "certified," or "guaranteed milk"—in feeding their babies, excepting in hot weather, when it is safer to assume the risk of scurvy developing than cholera infantum or entero-colitis. *Pasteurizing* is preferable to sterilizing, but even this destroys the germicidal action of raw milk which, according to Freudenthal, is one of its natural properties. Personally I prefer the use of raw pure milk in the colder months of the year.

\*The Milk Commission of the New York County Med. Soc. decided upon a standard to which the milk supplied to that city should conform, which is the following: Acidity must not exceed 2-10 of 1 per cent., there must be no more than 30,000 bacteria to the c. c., and butter fat must be present to the amount of at least  $3\frac{1}{2}$  per cent. To the dealers attaining this standard a certificate is to be issued and their milk known as "certified milk." (*Med. Record*, Vol. 60, No. 15.)

The Milk Commission of the Philadelphia Pediatric Society makes the following requirements: The specific gravity shall range from 1.029 to 1.034, reaction neutral or faintly acid; proteids, 3.5 to 4.5 per cent., sugar, 4 to 5 per cent., fat, 3.5 to 4.5 per cent. It must be free from all contaminated matter and from the addition of chemical substances and coloring matter. It must be free from pus and injurious germs and have no more than 10,000 germs of any kind to the c. c. (*Archives of Pediatrics*, March, 1902.)

**Pasteurizing** is practiced by immersing the bottle containing the milk into a receptacle holding water to the level of the milk in the bottle. The water in the receptacle is brought to the boiling-point; the bottle, stoppered with sterilized absorbent cotton, becomes highly heated in the boiling water, and the source of heat having been removed, the bottle is allowed to remain in the hot water forty-five minutes when it



FIG. 26.—FREEMAN PASTEURIZER.\*

is rapidly cooled under a jet of water and placed on ice. By this process the milk has been brought to a temperature of  $75^{\circ}\text{C}$ ., or  $167^{\circ}\text{F}$ ., and maintained at that heat for an average of half an hour, which is sufficient for all practical purposes. The Freeman Pasteurizer (Fig. 26) is an inexpensive apparatus and by its use much better results are obtained than by the makeshift method described. The Arnold Steam Sterilizer

---

\*Instructions for using Dr. Freeman's Apparatus for Low Temperature Sterilization of Milk by Pasteurization

1. Fill the pail to the level of the groove with water, cover it and put it on the stove to boil, the receptacle for the bottles having been left out
2. Fill the body of each bottle with milk or some modification of milk

(Fig. 27) is another good apparatus very convenient for home use, when sterilizing is to be carried out.

Pasteurizing milk does not render it sterile in the bacteriological sense; it however destroys the various saprophytic germs that are responsible for many cases of diarrhœas; the bacillus of tuberculosis, cholera, typhoid fever, and diphtheria, and the organism of scarlet fever. Spores are not destroyed, but as it requires several days' time for their development any that may be present will not prove a menace to the child, if the food is freshly prepared daily.

H. Lahmann (*"Allgem. Med. Central Zeitg.,"* lxx., 1896) believes that scurvy and rickets developing in infants fed on sterilized milk is due to the exclusive milk diet, and not to the process of sterilization. Milk contains too small a percentage of iron, soda, and lime, and to remedy this defect he adds fruit juices to the dietary. The juice of oranges, cherries, strawberries, and other fruits is recommended, and after the third month this can be given with impunity. The evil results from the use of sterilized milk are not seen in a day—they are the outcome of feeding over a prolonged time. There is therefore no objection to the use of sterilized milk during hot spells, while traveling, or when an excellent quality of milk cannot be obtained. One should, however, always endeavor to give the child raw milk whenever this is feasible.

In proper proportion for feeding, stopper with a wad of cotton batting and put in a refrigerator. If all the bottles which the receptacle holds are not needed fill the remaining cylinders with cold water. Each space in the receptacle must be filled.

3 When the water in the pail on the stove boils thoroughly, take the bottles of milk from the refrigerator and put them in the spaces for them in the receptacle

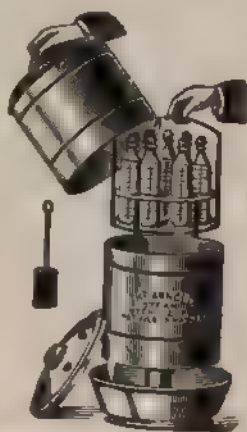


FIG. 27. ARNOLD STEAM STERILIZER.



If a good milk can be obtained twice daily it is rarely necessary to sterilize or pasteurize, excepting as before stated during hot spells, and also, when there is a tendency to diarrhoea.

Even the continued use of pasteurized milk may bring with it the appearance of signs of rickets and even scurvy. Sill (*N. Y. Med. Record*, Dec. 27, 1902) claims that in 97 per cent. of a long series of infants under his observation that were fed on pasteurized milk there were unmistakable signs of rickets or scurvy, or a combination of these diseases. On the other hand we occasionally see perfectly healthy infants that have been on pasteurized milk since birth.

---

4. Pour cold water into each of these spaces so as to surround the body of the bottle.

5. Take the pail of boiling water from the stove and put it on a table or mat. Do not put it on metal or stone. Be sure that the pail is still filled exactly to the level of the groove and that the water is boiling vigorously.

6. Set the receptacle containing the bottles of milk into the pail of boiling water, so that the wire (A) will rest on the support (C), cover the pail quickly and let it stand forty-five minutes. During this period the pail must not be on the stove and the cover must not be removed.

7. Now uncover the pail and lift the receptacle and turn it so that the wire (B) will rest on the support (C), thus elevating the top of the receptacle above that of the pail. Put the pail containing the receptacle elevated in this manner in a basin under a faucet to which a rubber pipe may be attached connecting it with the pail (Fig. 2). The water will overflow from the pail into the basin. Or the pail may be stood under a pump, fresh cold water being pumped into it every few minutes.

The above described method of cooling is the best. When, however, it is not possible to cool the milk in this way, the cooling may be accomplished by placing the receptacle containing the bottles of milk in iced water, or by simply standing the bottles on wood in a refrigerator.

8. To warm the milk for use, put the bottle containing it in a vessel of cold water on the stove, and leave it until it is warm. Use a fresh bottle for each feeding.

9. Wash the bottles thoroughly after using, and once a day put all the empty bottles in a kettle of cold water on the stove and let this water boil for an hour. The bottles should then be taken out and stood bottom up until used.

Milk sterilized by this apparatus may be used for food during the following twenty-four hours.

The Freeman Pasteurizer is manufactured by James T. Dougherty, 409 and 411 West 59th Street, New York City.

THE PREPARATION AND INDICATION FOR OTHER FOODS AND  
ADJUVANTS TO THE CHILD'S DIETARY.

## ARTIFICIAL FOODS.

**Barley-Water.**—This is a most useful adjuvant in the treatment of many conditions peculiar to infants. According to a series of experiments by Cautley, a weak barley-water will render the curds of milk, when precipitated by acetic acid, much finer than is the case with any other diluent. It is a bland, demulcent liquid, possessing some nutritive properties, mainly from the presence of starch. It should therefore be used cautiously until the amylotic functions of the saliva and pancreatic juice have been developed. Being demulcent, and containing a carbohydrate element which is not so favorable a medium for the development of micro-organisms as a proteid it is especially serviceable in the acute summer diarrhoeas of infants, either as an attenuant of the milk or when given pure. It is best made as follows: "Take two ounces of pearl barley and wash well with cold water, rejecting the washings. Afterwards boil with a pint and a half of water for twenty minutes in a covered vessel, and strain."—(PAVY.) A quicker method is to make it from barley flour, a tablespoonful to the quart, boiled fifteen minutes.

**Rice-Water.**—This is a very nutritious, soothing drink in acute intestinal troubles. "Thoroughly wash one ounce of rice with cold water. Then macerate for three hours in a quart of water kept at a tepid heat, and afterwards boil slowly for an hour, and strain."—(PAVY.)

**Rice-Paste.**—Dr. George B. Fowler ("N. Y. Med. Record," No. 12, 1890) highly recommends a paste, made by adding four tablespoonfuls of rice to three pints of water, boiling half an hour and then setting aside to simmer, water being occasionally added to maintain the three pints. This is strained through a colander and cooled, when a paste is formed. Three tablespoonfuls of the paste are added to half a pint of sterilized milk. Dr. Alonzo Barnes of this city has



had excellent results from this preparation in summer-complaint. Rice is perhaps the most readily assimilated starchy food.

**Oatmeal-Water.**—This is contraindicated in diarrhoeal affections, but is useful as a diluent in constipation. A tablespoonful of oatmeal is added to a pint of water and brought to the boiling-point under constant stirring. It is then set aside, allowed to cool, and strained.

**Albumen-Water.**—This is a highly nutritious, easily digested drink, and is often retained where the stomach rebels against more substantial forms of food. “Take the white of a fresh egg and cut it in various directions with a clean pair of scissors. Shake it up in a flask with a pinch of salt and six ounces of pure cold water. Strain through muslin.”

**Beef Tea.**—It is needless to mention here that beef tea contains no virtue beyond its stomachic and stimulating effects. It is useful in low, febrile conditions and where there is lack of reaction. In order to render it nutritious, beef pulp or a cereal must be added.

**Chicken- and Mutton-Broth.**—These broths are less stimulating than beef tea, but are better tolerated where there is much fever. Chicken broth contains some gelatin.

**Beef Juice.**—When properly prepared, this is a highly nutritious albuminous form of food. It is an excellent food in anæmia and where the digestive powers are weak, but lithæmic symptoms must be watched for when used over an extended period. To obtain the juice, a piece of sirloin steak, or any good piece of beef from which the fat and connective tissue have been removed, is quickly broiled in a hot pan, placed in a strong lemon squeezer, or, better still, the beef press, especially made for this purpose, and the juice squeezed out. It may be served warm with seasoning or on bread; also diluted with ice-water. Boiling coagulates the myosin and serum albumin, and renders the product less digestible.

**Junket** is often useful to vary the monotony of a milk

diet. It can be prepared with rennet or Fairchild's essence of pepsin.

**Peptonized Milk.**—This is a most satisfactory food in low typhoid states and sometimes in dyspeptic cases, used until the digestion has regained its normal condition. For rectal feeding it is extremely valuable. The milk used for this purpose must not be rich in cream.—(GILMAN THOMPSON.) The quickest and most satisfactory method of preparing it is to dissolve the contents of a Fairchild's peptonizing tube in four ounces of cool water, adding a pint of milk. The bottle containing the mixture is placed in hot water of a temperature that can be borne by the hand for a minute without discomfort (STARR), and allowed to remain thirty minutes. If this renders it too bitter, it should be removed earlier.

**Peptogenic Milk-Powder.**—This is a powder containing the pancreatic ferment and milk sugar. By adding it to a mixture of cream, milk and water, we obtain a modified milk, resembling human milk in composition and one in which the casein is at the same time partially predigested. It is well adapted to infants with weak or poorly developed digestive organs; I have seen it do good in premature and under-developed infants. A mixture of one-half pint milk, one-half pint water, four tablespoonfuls of cream and four measures of peptogenic milk powders makes a close imitation of human milk in composition. The process of peptonizing is essentially the same as in the case of the peptonizing tube above referred to. It can, therefore, be checked by bringing the milk to a boil or continued by keeping it warm (at 115° F.). As the child's digestion improves the peptonizing process should be shortened until the milk is tolerated in its natural state.

**Malt Diastase—Liebig's Food.**—Ground malt possesses marked diastatic properties, and when added to a starchy food, converts the latter into maltose. Malt extracts have the same power, but to a less degree. In amylaceous dyspepsia the child's farinaceous food, such as oatmeal, rice,

cracker-paps and flour soup, should be sweetened with a malt extract instead of cane sugar. It can also be given alone before meals. Liebig's food contains gluten—the proteid of wheat and barley—dextrin and maltose. It is prepared as follows: “Mix a half-ounce each of ground malt and wheat flour, seven and one-fourth grains of potassium bicarbonate, with one ounce of water and five ounces of sweet cow's milk. Warm slowly and stir until thick. Remove from fire, stirring for five minutes; replace over fire and remove when quite thick.”—(GILMAN THOMPSON.) This mixture becomes thin and sweet as the diastatic process becomes completed, when it is again boiled and strained. Fothergill is a great advocate of ground malt. He recommends it in addition with baked flour and hot milk. *Maltine* is a very stable article, and, beside being a digestive agent, is rich in phosphorus and other food elements. When a mild stimulant is indicated, a liquid preparation, such as *Hoff's*, is very useful.

**Baked Flour.**—Through the process of baking the starch-granules are burst, and some of the starch is converted into dextrin, making it, on the whole, more digestible. A water-cracker is a good example of baked flour, but it contains some lard, which is necessary in the process of manufacture. As most infant foods are deficient in fat, it is rather an advantage than otherwise; and if these crackers are rolled to a fine powder, stirred to a paste with cold water, and boiled with sufficient milk to make a thin pap, we have here a highly-nutritious food, easily digested by most babies after the sixth month. It can be sweetened with a malt preparation, which will prevent constipation. Comparing this food with the artificial foods flooding the market, we can see readily that the only advantage they have over simple home methods of preparing foods is the rapidity with which they are made and the saving of a little trouble. They are expensive, usually insufficiently nutritious, not always conforming to the formula advertised by the manufacturer, and, although they will save the mother a little trouble for the time being,

she will be fortunate, indeed, if the expense and worry attached to the development of tuberculosis, scurvy and rickets be spared her at a later date.

**Fruit Juices.**—In a previous chapter the necessity of using fruit juices where there is a tendency to scurvy and rickets was pointed out. Where fresh fruit cannot be obtained, the sweetened juice of dried plums, apples, apricots and the like can be used. In constipation they are often called for.

**Fat—Cod-Liver Oil.**—It may be that fat has been insufficiently supplied in the child's dietary, or that the child cannot properly digest and assimilate it. In the latter case cod-liver oil often comes to the rescue. It is best given as recommended by Fothergill, *i. e.*, taken about an hour after eating, when the food passes out of the stomach into the duodenum. In this way it does not needlessly provoke the stomach, and the disagreeable eructations are avoided. Sometimes an emulsion acts better than the pure oil.

The marrow from a shin-bone spread on bread while hot, and a little salt added, is an excellent food for anæmic children. Fat is the necessary food in struma and rickets. Butter-taffy is a pleasant way of supplying fat when cod-liver oil is refused, and is highly praised by Fothergill.

**Stimulants.**—Brandy, well diluted, is the best alcoholic stimulant. Beginning with half an ounce, the quantity can be increased to one ounce in twenty-four hours for a child one year old. I have often substituted alcohol sponge-baths (one part of alcohol to three parts water), and a compress of dilute alcohol applied to the abdomen, for the internal administration of alcohol, with entire satisfaction. Eggnog is an excellent stimulant and food in convalescence from acute illnesses; it is also valuable in the debility of phthisis. Malt extracts have been referred to.

**Artificial Foods.**—Any one who has taken the trouble to acquaint himself with the method of modifying cow's milk to resemble human milk in composition, and has observed the

results obtained from this method of feeding, and also has studied the simple method of preparing suitable articles of diet for the child in health and in disease, as detailed above, must fail to see any special necessity for the numerous proprietary foods so extensively used and advertised. And yet there is a field for them ; there are times when it is extremely convenient to have an article at command requiring simply the addition of hot water or milk for its preparation, and at the same time know that we can rely on it and get results. The mistake is to use a prepared food over a prolonged period of time, for then the mischief is done, but if we employ these foods with judgment, they are very useful. For example, *Horlick's Malted Milk* is often retained when other food is vomited ; it requires simply the addition of boiling water in its preparation, and will sustain life for a long period of time. It is, therefore, an excellent food to be used in travelling and in some acute conditions. A cup of hot malted milk at bedtime is conducive to restful sleep, this action being usually very marked and to be relied upon, more so than from the use of plain milk, no doubt owing to its greater digestibility.

**Condensed Milk** contains too much sugar and too little fat to be a suitable infant's food, being only permissible in case of emergency. Evaporated milk, without the addition of sugar, prepared from a milk rich in fat, is a much better substitute for fresh milk.

**Mellin's Food** is a Liebig Food and can be used when it is not convenient to prepare the Liebig food at home. It is useful in constipation, and is very fattening. When made according to directions it closely resembles mother's milk, but it must be remembered that the cow's milk which is added to this food is the main factor in the formula.

The **Allenbury's Foods** are excellent artificial foods made in a series of three formulæ, the composition of each aiming to correspond to the needs of the different periods of infancy. The milk food, No. 1, is to be used during the first three

months of infancy. It contains casein, fat and sugar in the correct proportion required for the digestive powers at this age. Food No. 2 is similar to the No. 1, but contains in addition a certain amount of maltose, dextrose, soluble phosphates and albuminoids. It is intended for the third to sixth month. The food No. 3 is essentially a farinaceous food, requiring the addition of cow's milk in its preparation. It is intended for children of six months and upward. The manufacturers of these foods, willing to admit that scurvy and rickets are likely to occur in children fed exclusively upon artificial foods over an extended period of time (to say nothing of malnutrition), wisely suggest the daily use of a dessert-spoonful of grape or orange juice to be given two or three times a week after the third month, and later on also raw meat juice.

Artificial foods, therefore, have their place ; they are never absolutely necessary, only being convenient contrivances of our progressive age, and they can never supplant mother's milk or cow's milk modified by strictly scientific methods.

## CHAPTER V.

### DISEASES OF THE NEW-BORN.

A variety of pathological conditions is to be observed in the new-born, resulting either from mechanical injury or from infection. Certain physiological changes taking place in the organism may also give rise to disturbances peculiar to this period of life; these are notably asphyxia, cyanosis, and icterus.

#### ASPHYXIA.

Asphyxia of the new-born may be either of intra- or extra-uterine origin. *Intra-uterine asphyxia* results from the interruption of the placental circulation through compression of the cord or premature separation of the placenta. Respiratory efforts are excited in the child through the resulting carbonization of the blood and the lungs consequently become filled with amniotic fluid.

*Extra-uterine asphyxia* presents itself immediately on or a short time after birth. The degree of asphyxia may be of several grades, varying from a simple interference with the respiratory function from the collection of mucus or other foreign substances in the pharynx and trachea to complete cessation of respiration. In the latter case the child may be robust when born and present all the signs of active asphyxia, the body surface being cyanotic and the face bloated (sthenic asphyxia); or it may be pallid and limp and apparently lifeless (asthenic asphyxia). A frequent cause of the asthenic form is *pial hæmorrhage*, the irritability of the respiratory centres being abolished through the intra-cranial pressure. In the absence of hæmorrhage, malformations of the respiratory or circulatory organs, pulmonary atelectasis, pulmonary syphilis, pneumonia or premature birth may be mentioned as causes.

The results of asphyxia are stagnation of dark, fluid blood in the veins and filling of the right ventricle, hyperæmia of the various organs, and petechial hæmorrhages.

The reflexes are not abolished in the sthenic variety and the pulse is slow but perceptible. It presents a better prognosis than the asthenic variety, in which there is pallor of the body surface, abolition of reflexes, and imperceptible pulse.

The *treatment* consists in the removal of all obstruction such as mucus and amniotic fluid from the air-passages, supplemented by measures calculated to set up respiratory efforts through peripheral irritation. The alternate warm and cold bath is very efficacious. In the asthenic variety the warm bath alone should be employed, together with artificial respiration, but when the asphyxia is only a symptom of one of the serious conditions above enumerated, the prognosis is utterly hopeless.

#### CEPHALEMATOMA.

A cepheleatoma is a tumefaction situated upon one of the cranial bones, usually the parietal, caused by hæmorrhage beneath the periosteum. It results from injury sustained during parturition, and is frequently encountered in children born through a narrow pelvis. Being entirely external no symptoms are induced thereby, the clot becoming organized and absorbed in the course of several weeks. Usually it does not become manifest until a few days after birth and may be confounded with hernia cerebri, but the latter is most frequently situated either at the root of the nose or the nape of the neck, and presents a distinct bony edge, indicating the opening from which the sac protrudes. No treatment is required.

#### HEMATOMA OF STERNO-MASTOID MUSCLE.

This usually affects the belly of the right sterno-mastoid muscle, most commonly appearing after breach labors, being



the result of twisting of the head during parturition. A firm elastic, egg-shaped swelling appears in the middle of the muscle about two weeks after birth and is accompanied by torticollis. It disappears in the course of a few weeks and requires no treatment, excepting such measures as will hasten absorption, namely, hot fomentations and *Arnica* internally.

#### INTRACRANIAL HÆMORRHAGES.

Apoplexy of the new-born is encountered as a venous or capillary hæmorrhage of the meninges of the brain, less frequently taking place into the cortex. It results from direct injury sustained during birth. This condition is fully discussed under cerebral palsies. Other forms of injury to the nervous system encountered at this period are facial and brachial paralyses, resulting from pressure or traction upon the nerve trunks supplying these parts.

#### SEPTIC AND OTHER INFECTIONS IN THE NEW-BORN.

Septic infection in the majority of instances takes place through the umbilicus. There may, however, be an intra-uterine infection through the placenta or by the aspiration of amniotic fluid containing pathogenic micro-organisms. Again, an abrasion of the skin or of the mucous membranes may give entrance into the system of germs. When the port of entrance cannot be discovered the infection is spoken of as "cryptogenic."

The *pathological findings* depend upon the mode of infection. Often it is impossible to find the site at which infection took place and the evidences of *septicæmia* alone exist. There is fever; rapid and shallow respiration, vomiting, diarrhoea and wasting. Collapse with a fatal termination is the usual outcome. Symptomatic icterus and internal hæmorrhages are associated conditions.

1. In the cases in which infection takes place through the respiratory tract, the evidences of septic pneumonia, frequently with bloody extravasations into the pericardium and pleura, are found.

2. *Infection through the umbilicus* gives rise to either a local or general sepsis. Under the heading of the former are included *umbilical arteritis*, *phlebitis* and *omphalitis*. In *omphalitis* there is an involvement of the surrounding cellular tissues and suppuration results. It occurs most frequently during the second and third weeks. The prognosis is good under prompt surgical treatment, but extension to the peritoneum with general sepsis may occur.

*General sepsis* originating in infection through the umbilicus is almost invariably associated with *peritonitis*. Besides this localization there may also occur septic pleuropneumonia; pericarditis; meningitis; gastro-enteritis; osteomyelitis and arthritis. The most frequent of these conditions is *peritonitis*; the next in frequency being pneumonia, then pleurisy; meningitis; meningeal hæmorrhage; entero-colitis; pericarditis and meningeal hæmorrhage, in the order as named (Bednar).

3. There are also a number of infectious conditions not originating in the umbilicus. They are as follows:

**Erysipelas.**—The distinct type of cellulitis resulting from infection with the *streptococcus pyogenes* is occasionally encountered in the new-born. An abrasion of the skin or mucous membrane is the usual site of infection, although it may originate in the umbilicus. In the latter instance a fatal termination is the rule.

The *remedies* most useful are *Belladonna*, *Apis*, *Rhus tox.*, and *Graphites*. Locally a 10 per cent. aqueous solution of Ichthyol proves a valuable adjuvant. Painting the border of the affected area with Collodion to check its spread has not proven of much value in my hands. In the severe cases complicated with *omphalitis* or *phlegmon* the stronger antiseptics must be applied locally.

**Tetanus.**—The bacillus of tetanus may be inoculated at the site of an abrasion of the skin or of a mucous membrane, or it may gain entrance through the cord. Infection at the umbilicus usually occurs at the time of the separation of the stump of the cord.

The symptoms are identical with those observed in the adult, the earliest manifestation being rigidity of the jaws, occurring as stated above, shortly after the separation of the cord-stump. This trismus is followed by tonic spasms of the muscles of the neck and extremities, occurring paroxysmally. As a rule, it terminates fatally within a few days, although it may pursue a protracted course and result in recovery.

The disease is by no means as frequently encountered now as it was in the pre-antiseptic days, when no precautions were taken in the dressing of the cord and when the granulating surface left after the separation of the stump was not protected against the invasion of germs.

The *treatment* is both local and internal. The site of infection should be dressed with gauze wrung from a one in two thousand solution of the bichloride of mercury in order to check the further progress of infection, and if there be a focus of suppuration, free drainage must be instituted. *Hypericum*  $\theta$  may be administered with the hope of influencing the course of the disease. Other remedies that have been recommended are *Belladonna*, *Cicuta*, *Hydrocyanic acid*, *Lachesis*, *Nux vomica*, *Physostigma* and *Stramonium*.

Tetanus *Antitoxin*, although a true antitoxin, has not as yet displayed a perceptible advantage over other methods of treatment, nevertheless its use should not be omitted. As the poison of tetanus is an intracellular toxin, only the very early use of the serum offers any hope of cure. In order to relieve suffering, if our remedies fail to act favorably, *Chloral hydrate* in one-half grain doses should be administered. Fifteen grains daily may be used. When given per rectum twice that amount is necessary. Hot bottles should also be tried, as they enhance the action of the drug and give much relief. The narcotics are of less value.

#### ACUTE FATTY DEGENERATION, OR BUHL'S DISEASE.

This disease was first described by Buhl in 1860, and presents parenchymatous inflammation, fatty degeneration and

hæmorrhages in the heart, liver and lungs. It is probably of infectious origin. It is rare, and is only seen in lying-in hospitals. The children are usually born asphyxiated, and they do not entirely recover from this state. Cyanosis supervenes, and they either die at this time, or the course of the disease is protracted, and bloody diarrhoea, hæmorrhage from the navel, mouth, nose and conjunctiva, and icterus, set in. Later, œdema of the skin occurs, and death from collapse follows at about the end of the second week. The diagnosis can only be positively made by a microscopic examination of the organs. The course is always fatal.

#### ACUTE HÆMOGLOBINURIA, OR WINKEL'S DISEASE.

In 1879 Winkel encountered a series of twenty-three cases of hæmoglobinuria occurring in the new-born, associated with cyanosis, icterus, and hæmorrhages in the various organs, with a fatal termination within thirty-two hours in the average of cases. The cause is unknown, but it is undoubtedly an infection. Other cases have been reported, but not in such an extensive epidemic as the above.—(WINKEL, *Lehrbuch der Geburtshülfe*.) Hamill and Nicholson in a series of carefully studied infections in the new-born (*Archives of Pediatrics*, Sept., 1903) have found that a variety of micro-organisms is to be encountered, showing that careless nursing is most likely at the bottom of these infections. They would include Winkel's disease, Buhl's disease and melena among the acute infections of the new-born, although hæmorrhagic conditions at this time of life may also be the result of syphilis, asphyxia, trauma and malformations.

#### OPHTHALMIA NEONATORUM.

The violent conjunctivitis of the new-born, which at times results in destruction of the entire eye is due to infection with the gonococcus of Neisser. When the infant is infected during parturition, the symptoms make their appearance on the third or fourth day; in some instances the eyes are

probably infected later and symptoms do not arise until a week or more.

The first indication of the trouble is redness and swelling of the palpebral and ocular conjunctiva, puffiness of the eye-lids and catarrhal secretion. The secretion rapidly becomes purulent and the eye-lids infiltrated and leathery. In virulent cases chemosis is pronounced and the cornea is deprived of its nutrition through compression of the blood vessels at the sclero-corneal margin. The cornea becomes opaque, its epithelium is desquamated and perforation may result.

A benign, non-gonorrhoeal form is also encountered. This is recognized by its mild course and by the microscopic appearance of the secretion which contains the ordinary pyogenic organisms.

The *prognosis* must always be guarded; it is especially unfavorable in cases that have progressed before treatment is instituted. It is claimed that from 25 to 30 per cent. of blindness can be accredited to ophthalmia neonatorum.

*Treatment.*—On the first indication of ophthalmia the eyes should be irrigated hourly with a 2 per cent. solution of *Boric acid* and covered with compresses wrung from ice water or laid on a cake of ice and kept constantly applied and changed when they become soiled and warm. As soon as the discharge becomes thick and creamy, a few drops of a solution of *Nitrate of Silver*, three to four grains to the ounce should be instilled into the eyes two to three times daily. At the same time, as the discharge increases, it is better to resort to frequent irrigation of the eyes with warm *Boric acid* solution every twenty minutes if necessary, and discontinue the compresses. A bad case will engage the entire attention of two nurses, one for the day and the other for the night. With the subsidence in the inflammation and when the eye-lids lose their infiltrated character, a few drops of a 4 per cent. solution of *Nitrate of Silver* should be dropped upon their everted surface, taking care not to allow it to run into the eye. This may be followed by irrigating with normal salt solution.

In order to satisfactorily inspect the cornea from day to day and to properly flush out the conjunctival sacs, it is well to make use of retractors. Should *Nitrate of silver* appear too irritating, *Protargol* in a 1 or 2 per cent. solution may be substituted.

When the cornea becomes involved a drop of a 1 per cent. solution of *Atropine sulph.* must be instilled twice daily. In threatening perforation, *Eserine* may be tried. Internally, *Aconite* in the early stages; *Arg. nit.* later. The responsibility of these cases is so great that an oculist should always be taken in consultation.

#### MASTITIS.

Inflammation of the mammæ with abscess formation is a common result of squeezing out the breasts in a rough manner. In the new-born there is frequently present a cholestum-like secretion and any form of mechanical irritation of such a breast is likely to result in inflammation and suppuration. Under the use of hot fomentations and the administration of *Belladonna* or *Bryonia* according as the symptoms of either of these predominate, followed by *Hepar*, resolution promptly results.

#### ICTERUS NEONATORUM.

Icterus may occur *symptomatically* as a hæmatogenous jaundice in septicæmia, Buhl's disease and Winkel's disease, or it may be due to congenital or syphilitic stricture of the hepatic duct.

A *physiological* icterus occurring several days after birth, disappearing spontaneously in the course of a week, is observed in from 79 to 84 per cent. of all infants (PORAK, CRUSE). It most frequently occurs when birth has been premature, or if ligation of the cord has been delayed. According to Birch-Hirschfeld, swelling of the capsule of Glisson takes place from interruption of the circulation in the umbilical vein, with resulting pressure upon the biliary ducts

and hepatogenous jaundice. Hofmeier is of the opinion that the icterus is hæmatogenous in origin, depending upon an extensive destruction of red blood corpuscles, a process which takes place in the liver most actively at this period of life. Stadelmann positively denies the existence of hæmatogenous icterus and he claims that the pigment found in the urine in pernicious anæmia, malaria and acute yellow atrophy of the liver is urobilin and not bilirubin. The concensus of opinion however seems to be favorable to the view that in icterus neonatorum there are two factors active, namely, fall of blood pressure in the hepatic circulation so that the pressure in the bile ducts becomes greater than in the hepatic veins, and excessive destruction of red corpuscles, making it impossible for the liver to transform all the pigment into bilirubin.

#### ŒDEMA.

In delicate, feeble infants during early life, a general œdema, affecting at first the eyelids and the dorsum of the hands and feet, and if it progresses, involving the entire cutaneous surface, may develop as a result of a feeble heart muscle. The kidneys are normal in these cases. Ascites seldom occurs. It has been suggested that some toxic agent, probably of gastro-intestinal origin may affect the lymphatics and thus set up the œdema. *Kali carb.* seems the best indicated remedy.

#### GASTRO-INTESTINAL HÆMORRHAGE, OR MELENA.

Hæmorrhage from the stomach and bowels may take place shortly after birth, and terminate fatally within a few days. These hæmorrhages may result from congestion and slight erosion of the mucous membrane of the lower bowel (as a result of thrombosis of the umbilical blood vessels or asphyxia), follicular ulceration of the stomach and intestines, or from a round, perforating ulcer, and also from any of the infections above mentioned, beside constitutional diseases.



The possibility of follicular ulceration of the stomach and bowels existing in infants who have died suddenly without having displayed any of the symptoms of melena, either in the vomiting of blood or the passing of bloody stools, has been impressed upon me on several occasions by post-mortem findings. The pathology of this condition is more fully described under the diseases of the stomach. Etiologically these hæmorrhages undoubtedly belong to the infections of the new-born. The distinctive symptoms, bloody vomitus and bloody stools would indicate such remedies as *Hamelis* (abdomen sore to touch, hæmorrhage profuse, dark, or clots mixed with mucus) *Merc. cor.* (tenesmus; bright blood) *Argentum nitr.* (ulceration of stomach) *Arsenicum* (great prostration; septic cases). Other remedies may be suggested by the child's general condition. Supra-renal extract in one-half grain doses is the most satisfactory hemostatic in gastric hæmorrhage.

## GONORRHŒA.

The most common form of infection is of the eyes. Baginsky has recently reported the case of a male new-born in which genital gonorrhœa developed. Of late years frequent attention has been called to the prevalence of gonorrhœal arthritis in infants. There is no doubt that the majority of cases of acute arthritis in infants is gonorrhœal. General septic infection with polyarthritis and constitutional symptoms occur. I have recently had such a case in which Dr. Sappington was able to demonstrate the gonococcus in the pus from the joints. The course was protracted and the infant died from exhaustion. The arthritis was preceded by gonorrhœal ophthalmia. Kimball (*N. Y. Record*, Nov. 14, 1903) has reported eight cases of gonorrhœal pyæmia in infants. In none of his cases was a local infection demonstrable. These cases proved fatal. The temperature is irregular and usually runs high. Many joints may be affected, even the fingers and toes, and the fusiform swelling of a finger



may lead us to suspect tuberculosis or syphilitic dactylitis if we are not on our guard. The polyarthritides however eliminates these conditions. Other foci of pus are found scattered throughout the body.

#### SUDDEN DEATH IN INFANTS.

Sudden death in the new-born is most frequently due to visceral hæmorrhages resulting from compression of the head during birth or from hæmorrhage into the internal organs. The latter is more frequent in breach cases, no doubt as a result of improperly made traction.

Malformations of the viscera, either demonstrable or unsuspected, are common causes of sudden death in young infants. Here may be discussed **thymic death**, a subject in which renewed interest has been shown only in recent years. While Paltauf denies that pressure from the thymus plays a role in the sudden death of these infants, attributing it to the clinical entity he has termed *status lymphaticus*, or *lymphatism* (see Constitutional Diseases), still the theory that thymic death can occur has many adherents, notably in Jacobi. The latter writes: "It [the *thymus* gland] is largest, normally, from the third to the twentieth month; about the ninth month it was found, in usual instances, from 1.5 to 2 centimetres in thickness. As the distance between the manubrium sterni and the vertebral column is but two centimetres about the eighth month of life, the slightest increase of an enlarged thymus through distended circulation, by crying or otherwise, may prove suddenly fatal; for besides the thymus, the œsophagus, the trachea, the blood vessels, and the sympathetic and pneumogastric nerves are located in that narrow space. Bending the head backward during tracheotomy proved fatal. Swelling of the thymus in a cold bath may be dangerous" (*Therapeutics of Infancy and Childhood*). In discussing a case recently reported by Caille (*Archives of Pediatrics*, March, 1903) Jacobi called attention to the fact that but a few of the instances are on record since Kopp reported his first case of

thymic asthma nearly a hundred years ago. He related a case operated upon by König in which the gland was partly excised with life-saving results. For detailed report on this subject the reader is referred to Jacobi's monograph (Trans. Ass. of American Phys., Vol. III.) and to Friedjung's article (*Archive für Kinderheilk.*, Vol. 29).

**Atelectasis.**—This is either congenital or acquired. Complete atelectasis is seen in *asphyxia neonatorum*. In feeble infants atelectasis may develop after the lungs have been functioning, and if progressive it results in death. It is simply a manifestation of a general lack of resistance in the infant to its environment. During the course of bronchitis or broncho-pneumonia areas of atelectasis develop from the occlusion of the finer bronchial tubes. In some cases of marasmus nothing is found post-mortem excepting pulmonary atelectasis.

*Asphyxia* from the aspiration of food into the larynx is at times found to be the cause of sudden death in feeble infants. Sudden death may arise from laryngismus stridulus or in general convulsions, the determining cause being asphyxia. A retro-pharyngeal abscess or the pressure of tuberculosis bronchial glands upon the pneumogastric nerves or trachea may likewise cause sudden death.

*Sudden death after a few hours of illness with high temperature* is as a rule due to congestive pneumonia (HOLT). An infant several days old dying suddenly with high temperature and rapid respirations, at the Hahnemann Maternity during Dr. Korndoerfer's service, showed at autopsy a large hæmorrhage from the right middle meningeal artery following forceps delivery. The chief interest in the case rested in the utter absence of cerebral manifestations.

Sudden death may occur in the first twenty-four hours of a malignant scarlet fever before the eruption has made its appearance.

## CHAPTER VI.

### DISEASES OF THE MOUTH.

#### DENTITION.

The period of dentition represents the time during which the milk teeth make their appearance, and extends normally from the seventh month to the second year. The period of second dentition begins with the sixth year and is usually completed before puberty, with the exception of the wisdom teeth, which may appear as late as the twenty-first year. The term "teething" applies to the first dentition period, and embraces the various disturbances occurring at this time, when they can be directly traced to the teething process.

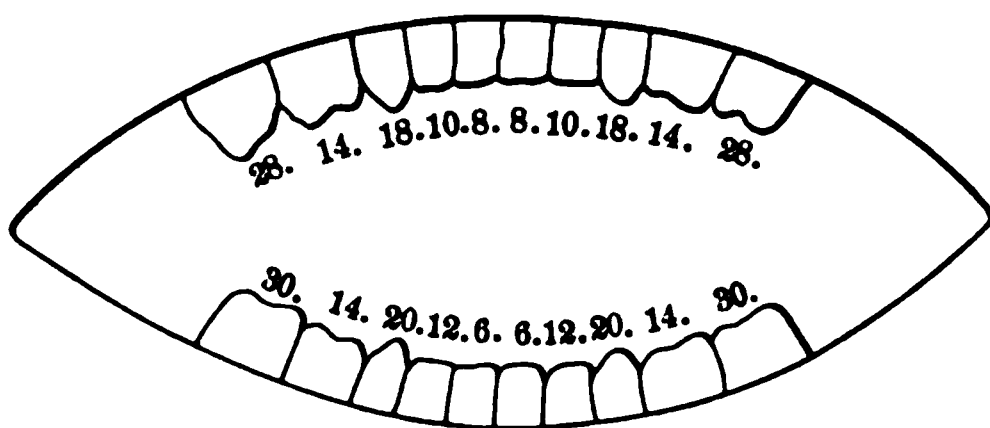


FIG. 28.—DIAGRAM SHOWING TIME OF ERUPTION OF THE MILK TEETH. (CHAPIN.)

Six to eight months after birth the two lower central incisors should make their appearance; the upper central incisors are usually a month later. The upper lateral incisors are the next in order, and at the end of a year the upper anterior molars should appear. At the fourteenth month the lower lateral incisors erupt, followed by the lower anterior molars.

The canine teeth appear between the sixteenth and twentieth months, and at the end of the second year the posterior molars are added to complete the set.

Soon after the eruption of the milk teeth absorption begins, commencing at the apex of the root and extending to the crown, so that they are either lost by an accidental tearing of the membranous attachment to the gums, or are displaced by the advancing permanent teeth.

Dentition is a purely physiological process, and should therefore run a normal, uneventful course. This is, however, unfortunately not always the case, and those who deny the possibility of a pathological condition arising from difficult or abnormal dentition do so simply from an unwillingness to recognize the relation between cause and effect so manifest in these cases. It is true much harm has been done by the lazy routine of attributing every ailment of infancy to "teething," or, finding the child in the act of cutting a tooth, neglecting to investigate further into the case, whereby many a pneumonia, meningitis, gastro-enteritis or other serious condition has been overlooked, and another child sent "over the hill to the cemetery." But, notwithstanding all this, teething is responsible for many disturbances, either directly or indirectly. It is hardly necessary to refer to the swollen, sensitive gums, the salivation, anorexia, irritability and slight fever, especially when several teeth are making their appearance at the same time, and when the gums are abnormally tough. Although lancing of the gums is to be condemned generally, still when the cusps of the molars are distinctly advanced beneath the mucous membrane and only delayed in making their appearance by the resisting state of the same, the use of the lance is imperative and will give immediate relief.

The disturbances induced are mostly reflex, although the changes occurring in the shape of the lower jaw have a direct influence upon the floor of the middle ear. Through the chorda tympani nerve the buccal cavity is brought into close communication with the faucial extremity of the Eustachian tube and the middle ear. Cooper (*Clinical Lectures Upon Inflammations of the Middle Ear*) remarks upon the frequency with which children develop a discharge

from the ear every time they cut a tooth. A common cause for an attack of otitis occurring during teething is extension of the inflammatory process in the mouth, *i. e.*, the catarrhal stomatitis, into the Eustachian tube to the middle ear, through continuity of structure.

A later event to be encountered is the decay of the teeth. The relationship of enlarged tonsils, cervical adenitis, dyspepsia, and other affections to carious teeth, is too well established to merit special discussion. It is also important that the milk teeth be preserved as long as possible, for their premature loss interferes with the full growth and proper development of the jaw, thereby inviting a contracted palate, abnormally small jaw or irregularities in the permanent teeth.

**Treatment.**—The hygiene of the mouth is of prime importance in preventing and ameliorating the local manifestations. Cleansing the gums with a soft linen cloth dipped in plain cold water is beneficial when they are hot and swollen. An occasional drink of cold water will serve the double purpose of cooling the gums when there is gingivitis and mitigating the febrile phenomena. One of the following remedies will be found efficient in meeting the local and general symptoms :

*Bell.* and *Cham.* are perhaps the most frequently employed remedies in teething disturbances, *Chamomilla* being indicated by the irritable temper, the greenish, offensive diarrhoea, and circumscribed redness of the cheeks, and *Belladonna* when there is cerebral hyperæmia, high fever, and tendency to convulsions. The gums are red and swollen.

*Ferrum phos.* is especially useful when the respiratory tract becomes involved, indicated by rapid breathing, hard, dry cough, hoarseness, great restlessness. *Acon.* is similar, but there is more nervous erethism and vascular excitement.

*Terebinthina.* This remedy was first recommended by Cooper, and it has proven of value in my hands. When there is great restlessness at night; interstitial distention of the gums; symptoms of intestinal irritation, such as starting and

twitching during sleep, gritting of the teeth and picking the nose, *Terebinthina* is strongly indicated and acts promptly.

The *Calcareas* are very useful, especially as constitutional remedies. The *Carbonate* is indicated in children teething rapidly, and follows well after *Belladonna* in acute conditions. The *Phosphate* is more suited to emaciated or rachitic infants with open fontanelles, delayed dentition and tendency to early decay of the teeth.

#### ABNORMALITIES OF THE TEETH.

The most characteristic deformity seen in the teeth is the condition first described by Hutchinson. As a result of congenital syphilis, a lack of development in the permanent teeth takes place, and, the enamel being deficient upon the cutting



FIG. 29.—HUTCHINSON TEETH. THE UPPER INCISORS SHOW INCOMPLETE NOTCHES. THEY DIVERGE, LEAVING A WIDE INTER-SPACE. AFTER HUTCHINSON.  
(BARTLETT'S DIAGNOSIS.)

surface of the upper central incisors, a semilunar notch is worn into them (Fig. 29). These teeth are shorter than normal and their sides somewhat sloping, giving them the form of a screwdriver, being narrower at their cutting edge than at the root. Besides, the canine teeth are rudimentary and peg-shaped. This combination of abnormal teeth is named after Hutchinson and considered pathognomonic of congenital syphilis, although not commonly encountered, even in syphilitic children.

The milk teeth are not characteristically affected by syphilis; they may be poor in quality and decay early, or they

may show irregularities in form and in their enamel covering. Any form of *stomatitis*, however, can affect the development of the teeth.

*Rickets* delays the eruption of the teeth, and in such children they are, as a rule, abnormally soft and decay early. The permanent teeth may show transverse ridges or a serrated edge as a result of rickets or stomatitis. Any disease affecting the general nutrition naturally shows its influence upon the teeth if it be active at the time of their eruption.

#### STOMATITIS.

The term stomatitis is applied to the several forms of inflammatory affections involving the mucous membrane of the buccal cavity. It is a common affection among children, and can be traced to various causes, each of which will be fully discussed under its appropriate heading.

#### CATARRHAL STOMATITIS.

This form of stomatitis presents an acute diffuse inflammation of the mucous membrane of the mouth.

**Etiology.**—The exciting cause is usually some form of local irritation, such as unclean nipples; improper food, or giving the food too hot; infection of the mouth with various microorganisms, made possible by lack of proper cleanliness. The predisposing factor is, in the vast majority of cases, gastrointestinal derangement. Hot weather and artificial feeding are therefore responsible for most cases of stomatitis. The scrofulous diathesis, with its tendency to catarrhal inflammations of all mucous membranes, is often present, and in such cases difficult dentition may be the exciting cause. It may be associated with the eruption of the teeth, or occur during an infectious disease.

**Symptomatology.**—Primarily there is heat and dryness of the mucous membrane of the mouth and gums, together with redness and swelling. This is generally uniform, although it may be more marked in circumscribed areas. Later there is

increased secretion of mucus and saliva, which generally dribbles from the mouth. Pain is present, and the pathognomonic symptom, "The child seizes the nipple eagerly, but after a few pulls at the breast drops it with a cry," is explained by this exquisite tenderness of the mouth. The child is fretful and feverish, and, owing to the inability to nurse successfully, soon loses in weight. Diarrhoea and vomiting must rather be considered as concomitants of the general condition than as a direct result of the stomatitis. It is usually of short duration and does not terminate in ulceration.

#### PITYRIASIS LINGUÆ

Is a chronic catarrhal inflammation involving the upper surface of the tongue, resulting in the characteristic condition known as *linguæ geographica*. It begins as a circular patch or patches of epithelial hyperplasia forming elevated whitish spots, which enlarge and ultimately begin to desquamate in the centre, forming irregular plaques, with islands of normal mucous membrane interspersed between the hyperplastic epithelium. Several of the ring-like lesions coalesce and form the geographical map giving the disease its name. This affection shows great tendency to recur, the interval between the disappearance of the old lesions and the reappearance of a new annular patch being usually of short duration. It is met with in children of all ages, in the healthy as well as sickly, although perhaps most frequently in the rachitic.

#### APHTHOUS STOMATITIS.

Aphthous stomatitis is a vesicular inflammation of the mucous membrane of the mouth, resulting in localized erosions.

**Etiology.**—The etiology of this affection is not well understood, unless it be considered as a more pronounced form of the catarrhal variety resulting in vesication. Forchheimer looks upon aphthous stomatitis as an acute infection of intestinal origin and he compares it to foot and mouth diseases



of cattle. Filatow believes it to be a local infection, as it often attacks several children in one family simultaneously. It is most commonly seen from the first to the third year.

**Pathology.**—Together with a diffuse catarrhal inflammation there is vesicle formation, destruction of the vesicle, and superficial erosion resulting at the site of the vesicle. These erosions usually have a yellowish or dirty-grayish base consisting of a fibrinous exudate and epithelial débris. The lesions are surrounded by a red areola, and several may coalesce, forming irregular superficial erosions. They heal by a skinning over of the epithelium from the periphery and leave no scars.

**Symptomatology.**—The general symptoms of stomatitis, together with the characteristic lesions described above, make up the clinical picture. There is more pain than in the catarrhal form. The lesions are most frequently located on the tongue, the inside of the lips and of the cheeks, and in some instances they are found on the palate and in the pharynx. The breath is not foul, as in ulcerative stomatitis, and the course is usually of short duration, although it may be self-prolonging by the interference with nutrition.

#### BEDNAR'S APHTHÆ.

This is a condition which must be distinguished from aphthous stomatitis, being only found in children between the ages of two days and six weeks, characterized by the formation of a round, superficial ulcer, one situated at each angle of the palate. The *prognosis* is usually favorable in this disease, although deep ulceration of the tissues has been observed. It is no doubt brought on by traumatism from the nipple or nurse's finger in washing the mouth.

#### APHTHÆ EPIZOOTICÆ.

This is an infectious form of vesicular stomatitis, resulting from the use of unsterilized milk from cows affected with the disease. There is more fever than in aphthous stomatitis,

salivation and coryza accompany the other symptoms, and the vesicles do not appear on the dorsum of the tongue or pharynx, but are usually situated on the soft palate, lips, gums and cheeks. There is also foetid breath, sometimes vomiting and diarrhoea. It runs its course in from one to two weeks.

In *varicella* vesicles often appear in the mouth, but they seldom break down, and the cutaneous manifestations are sufficient to differentiate it from aphthous stomatitis.

#### ULCERATIVE STOMATITIS; PUTRID SORE MOUTH.

This variety presents an inflammation of the mucous membrane of the mouth, accompanied by ulceration.

**Etiology.**—The destructive inflammation of ulcerative stomatitis is due to a local infection. Although it has occurred epidemically, no specific micro-organism has been demonstrated, and it seems that the ordinary pyogenic bacteria will induce the disease when they find a soil favorable to their propagation. We can, therefore, understand how lack of proper cleanliness of the mouth and an enfeebled constitution, with unhygienic surroundings and improper nourishment—perhaps a scorbutic state—will invite the outbreak of an attack of ulcerative stomatitis. It may also result from the abuse of the metallic drugs, notably *Mercury*, and never develops until *dentition* is well established.

**Pathology.**—The morbid process begins with an inflammation of the anterior border of the gums, at the roots of the teeth, most frequently on the lower jaw. Redness and swelling are the initial changes, after which a yellow line, indicating the beginning of the necrotic process, develops along the alveolar border and extends downwards. From the gums the process extends to the inner margin of the lips, and large ulcers are generally formed on the lining of the cheeks opposite to the molar teeth. The sides of the tongue frequently participate, becoming infected by direct contact with the lesions.

**Symptomatology.**—In the beginning of the disease there are the usual symptoms of stomatitis, but soon the characteristic foul breath develops, the pain becomes intense, and prostration and fever is more marked than in the other forms. This is easily understood when we consider the severity of the process and the intoxication resulting from the absorption of the putrid material forming in the mouth. Under proper treatment it may be eradicated in the course of a few days, although the *prognosis* must remain guarded in frail constitutions, or where the ulceration affects the deeper structures and there is general systemic involvement, as indicated by a continued high temperature; rapid, weak pulse, and lymphatic enlargement.

#### PARASITIC STOMATITIS; THRUSH.

Parasitic stomatitis is an affection of the mouth due to the development of a parasitic fungus within the mucous membrane, and is characterized by the appearance of milk-white patches which are difficult to remove and have a tendency to coalesce and spread extensively.

**Etiology.**—The *saccharomyces albicans*, a fungus of the group *saccharomyces*, is found in the mucous membrane wherever the lesions develop. If a portion of the white pellicle be removed and placed on a slide with a drop of liquor potassæ the mycelium and the spores can be readily made out. Plaut considers the ordinary mould fungus as the etiological factor.

Artificial feeding by careless methods, early life, exhausting diseases, catarrhal stomatitis, insufficient salivary secretion, unsanitary surroundings and lack of proper care are all prominent etiological factors. The disease can be communicated directly from one patient to another, and is quite common in foundling asylums and among the poorer classes.

**Pathology.**—The spores of the *saccharomyces albicans*, finding their way into the mouth of the infant, soon develop their mycelia, which penetrate the layers of the mucous

membrane and form the white patches or elevations so characteristic of the affection. These patches are difficult to remove, as they are within the mucous membrane, but there is no exudation or pus formation accompanying the process. The lesions usually begin as small white points on the inner surface of the cheeks, quickly spread and coalesce, so that in a short time the entire buccal cavity and pharynx may be involved. Extension to the œsophagus is rare, and to the stomach still rarer, as it confines itself almost exclusively to the squamous epithelium. Rare cases, however, are on record in which these localities were affected, beside the lower rectum, the female genitalia, the upper respiratory tract, intestines, and abraded cutaneous surfaces.

Preceding the outbreak of thrush the mucous membrane of the mouth is hot and dry; later there is a sticky mucous secretion, acid in reaction. This is partly due to a lack of the normal alkaline salivary secretion, and to saccharine fermentation, the result of the growth of the fungus.

**Symptomatology.**—Beside the objective symptoms already described there is generally a painful condition of the mouth, due to the catarrhal stomatitis set up by the fungus. Thrush being seldom a primary affection, as a rule developing during the course of some acute gastro-intestinal disorder or a more chronic exhaustive disease, it cannot be said to have many symptoms of its own, excepting the objective manifestations. The *prognosis*, therefore, depends upon the accompanying condition, and ordinarily it is very favorable; but in an enfeebled constitution the development of thrush is a most unfavorable sign, running a very stubborn course or indicating the hopelessness of the case.

**Diagnosis.**—The white pellicle of thrush closely resembles flakes of coagulated milk and in the beginning is often mistaken for such; but the difficulty with which these spots can be removed and the associated stomatitis readily differentiates it from such a condition. Thrush has been mistaken for diphtheritic deposit, but here the age of the patient, together with

the associated conditions, the absence of foul breath, glandular involvement, fever and prostration, and the superficial character of the lesions, should readily differentiate the two. Where doubt exists, the microscope should be resorted to.

#### GANGRENOUS STOMATITIS—NOMA.

A destructive inflammatory process involving usually the cheeks and developing secondarily to one of the exanthemata or to some exhausting disease.

**Etiology.**—It generally follows upon measles, scarlet fever, typhoid fever, or some form of exhausting disease, occurring most frequently between the age of three and six years and in the poorer classes. The pyogenic bacteria, notably the streptococcus pyogenes, are responsible for the destructive pathological changes. In a certain number of cases the diphtheria bacillus was present (WALSH).

**Pathology.**—Beginning on the inside of the cheek or near the corner of the mouth, a small vesicle, filled with a turbid fluid, is formed. The vesicle breaks and leaves a superficial ulcer with a hard, infiltrated base, which can be felt through the cheek. This breaks down and a rapidly spreading gangrenous process develops, with no tendency to limitation. The affected parts become infiltrated and œdematous, presenting a shiny, livid appearance.

**Symptomatology.**—Often the first symptom noticed will be the ulcer, as the vesicle is easily overlooked. The breath is foul, prostration profound, and the temperature of the septic-fever type. The *prognosis* is unfavorable, the patient either succumbing to septicæmia or to a secondary broncho-pneumonia; fatal hæmorrhage is rare. In the case of recovery there is usually marked deformity.

**Treatment of Stomatitis.**—All forms of stomatitis can, to a great measure, be prevented by strict attention to the hygiene of the mouth, as well as careful supervision of the diet and general hygiene of the child. With artificially-fed babies, it is important to have the nipples and bottles kept perfectly

clean and sterilized (see "Care of the Mouth," Chapter I). During the course of an acute illness, especially one of the infectious fevers, it is imperative to have the mouth kept in a perfectly clean and sweet condition, for it is in these cases that noma is liable to develop, particularly in the enfeebled and poorly nourished.

In the case of very young infants at the breast it is safer to wash the nipple with a Boric acid solution than to attempt to clean the babe's mouth. An injury to the mucous membrane is easily produced, which will act as the starting point of an infection.

Should stomatitis develop, a mild antiseptic wash will be sufficient to carry the case through, excepting in the gangrenous form, which is, strictly speaking, a surgical disease. For this purpose, either a 4 per cent. *Boric acid* solution, alcohol diluted with three parts water, or, in the ulcerative form, *Hydrogen dioxide*, one part to four of water, will be the least harmful and most serviceable antiseptic. In stubborn cases of thrush it may become necessary to touch the patches carefully with a 2 per cent. solution of *Silver nitrate*; this is to be followed by rinsing the mouth with salt water.

The *diet* is important in ulcerative stomatitis. By a restriction in the use of all salty articles of food, and the free use of fruit juices and vegetable broths, these cases recover more promptly than under ordinary treatment. Owing to the painful condition of the mouth the diet should be restricted to liquids and semi-solids, and in older children the use of a tube or feeding-cup with a spout will be very grateful.

*Borax* is perhaps the most useful remedy in the aphthous and parasitic form, especially in the early stages, with heat and dryness of the mouth. It may be applied directly to the affected parts either in pure form or in the first decimal trituration, which, being slightly sweet, is more pleasant to the child, or it may be used in the form of a saturated solution. The internal administration of *Borax* is also advantageous. The symptoms on which it is prescribed are:

Aphthæ ; vomiting ; flatulent distention of abdomen ; loose, yellowish, slimy stools ; greenish stools preceded by crying. Child cries out as if frightened during sleep.

*Mercurius* may be indicated in all forms, but pathologically it corresponds most closely to the ulcerative form. Personally I have obtained the best results from *Baptisia* in this variety, as the sphere of usefulness of *Mercury* seems limited to syphilitic cases. *Merc. corr.* 3x trit. is useful in bad cases of thrush. *Hepar* is the remedy for mercurial stomatitis. *Chlorate of Potash* in small doses is indicated in herpetic and aphthous stomatitis. Other remedies which may prove useful are :

*Arum triph.*—Aphthæ ; lips swollen.

*Æthusa.*—Thrush ; vomiting of large curds.

*Ars.*—Thrush ; exhausting diseases ; prostration ; dryness of mouth.

*Baptisia.*—Ulcerative stomatitis ; great fetor of breath ; offensive diarrhœa ; typhoid state. Also useful in mercurial stomatitis.

*Bry.*—Catarrhal stomatitis ; great dryness of mouth.

*Hydrastis.*—Superficial ulceration ; tenacious mucus.

*Natr. mur.*—Gums spongy ; superficial ulcers on tongue and cheeks.

*Nitr. ac.*—Ulcerative stomatitis ; after *Mercury* ; fetid breath and acrid saliva ; acrid diarrhœa ; cracking of the corners of the mouth.

*Rhus tox.*—Great restlessness ; saliva bloody ; lips cracked.

*Sulphur.*—Ulcerative stomatitis ; gums swollen and receding ; marasmus. Bright redness of lips.



## CHAPTER VII.

### DISEASES OF THE STOMACH.

The investigation of the diseases of the stomach in infants is based upon practically the same principles underlying this special department of clinical medicine in adults. Owing, however, to both anatomical and physiological differences, and I might add psychic, a slight divergence in methods must necessarily exist, the appreciation of which becomes of the most practical importance.

There are a number of nervous gastric disturbances—gastric neuroses—common in adults and, on the other hand, practically unknown in infancy. I refer to hyperchlorrhydria, idiopathic achylia gastrica, gastralgia, nausea and dyspepsia nervosa. This class represents states of sensory, motor and secretory irritation or depression and originates in causes not yet operating in infancy.

Again, malignant disease may practically be discarded from the category of gastric diseases in infants. Isolated cases have been reported, but the majority of these were never suspected during life.

The chief etiological factor in gastric disturbances in infants is *improper feeding*. This is a complex factor which may be analyzed into (a) the use of foods of improper composition; (b) the administration of abnormally large quantities of food; (c) irregularity in the time of feeding, and (d) improper temperature of the food. A last factor, so important that it merits separate discussion, is *infection*. The micro-organisms setting up pathological changes in the gastric mucosa almost invariably gain access to the system with the food. The prevention of such an infection is therefore entirely within our control.

The toxins generated by these micro-organisms act either



directly upon the mucous membrane or fermentative changes are induced in the chyme with the liberation of irritating products. Accordingly, bacterial contamination of the food is responsible for the occurrence of cholera infantum, ileocolitis, acute and subacute gastritis, and many cases of acute indigestion. The derangements resulting from improper foods are acute and chronic indigestion, gastric catarrh, gastric dilatation, colic and vomiting. *Colic* is a distressing symptom common in infants and is often gastric in origin. It is most often due to flatulency, although I am positive that a large number of cases depend upon the presence of tenacious mucus in the stomach, which acts by interfering with digestion and with the exit of the food through the pylorus.

Colic may also result from taking the food too rapidly, in too large quantities, and from excess of proteids. Again, some cases seem to depend upon an inability to digest fat in normal and even decreased percentage, owing to a gastrointestinal catarrh.

An important fact that cannot be disregarded is the impossibility of drawing a sharp line of demarcation between diseases of the stomach and intestines. In adults there is greater possibility of doing this. The infantile stomach is nothing more than a dilatation of the alimentary canal and is not completely differentiated from the same. Its position at first is almost vertical; its capacity is relatively small and its sphincters are immature. Physiologically it is also immature, the main work of digestion falling upon the intestinal tract. Under normal conditions, therefore, the food (breast-milk) is coagulated shortly after reaching the stomach by the rennin of the gastric juice. Hydrochloric acid is now secreted and the casein is converted into acid albumin (syntonin). The action of the pepsin simultaneously secreted is feeble. In fact, the food does not remain long enough in the stomach to be digested very completely. In a long series of cases that I have studied very critically I have found that in infants under six months there is rarely a trace of food in the stomach at

the end of an hour under normal circumstances. In fact, at any period of infancy the stomach should have emptied itself at the end of two hours. If gastric contents can be recovered after that time, we are confronted with an abnormal condition.

The various steps in the examination of the stomach are—

*Inspection.*—In an emaciated infant it is possible to see the outline of the lower border of the stomach when the same is distended. It is impossible to arbitrarily decide just where this should reach, because the stomach may undergo a certain amount of distension under normal conditions, and its size also varies in different individuals. A safe rule, however, to follow is to look upon any stomach as dilated that reaches below the umbilicus, unless there be an enteroptosis. The latter condition I have never encountered in an infant. Again, when the stomach is dilated its outline is abnormally large; the transverse position is exaggerated, the cardiac extremity showing the greatest amount of enlargement. Irregular or saccular dilatation is exceedingly rare.

A condition from which dilatation of the stomach must be differentiated is dilatation of the colon. The latter, however, presents a concave outline, while the lower border of the stomach is convex.

In pyloric obstruction, peristaltic waves beginning at the cardia and traveling toward the pylorus may be seen. Pyloric obstruction may be congenital or acquired. Some cases appear to be spasmodic in character. In typical cases the pyloric extremity of the stomach is found to be hypertrophied and sclerotic in nature at the autopsy. I believe a certain amount of pyloric obstruction not infrequently accompanies gastric catarrh, and I have been able to control the condition with systematically conducted lavage in a number of cases. Again, I have observed hyperacidity of the gastric contents in a few cases with gastric dilatation, presumably not due to over-feeding. It is fair to infer that spasm of the pylorus may result in these cases from irritation by the hyperacid gastric contents; it will require further investigation, however, to prove this assertion.

*Percussion.*—This is a most valuable aid in determining the size of the stomach. The best results are achieved by filling the stomach with water through a lavage apparatus and outlining the absolute dullness obtained in this way. I do not look upon this as a dangerous procedure when the child is placed in the prone position and the water poured in slowly, from only a moderate height (one to two feet).

Traube's semilunar space is not as sharply outlined in infants as in adults, owing to the vertical position of the stomach and the horizontal position of the ribs. It is difficult to elicit and throws no light on the present subjects.

*Mensuration.*—I will apply the term mensuration in the sense of estimating the capacity of the stomach. This can be done by pouring water into the stomach through a lavage apparatus from a graduate and noting the amount required to fill the stomach. If carried out as above specified there will be no danger of doing harm. The stomach of the new-born holds about one ounce, and its capacity increases at the rate of one ounce per month, so that a two months infant will have a capacity of three ounces; a three months infant, four ounces; four months, five ounces; five months, six ounces; six to eight months, seven ounces; eight to ten months, eight ounces; one year, nine ounces.

At the same time we can outline the stomach. These data give positive indications as to whether the stomach is normal or dilated. Another point, namely, the length of time the food remains in the stomach, also bears a strong relation to dilatation of the stomach. This will be discussed further on.

*Auscultation.*—Stenosis of the cardiac orifice is indicated by absence of the deglutition sound over the stomach. In dilatation splashing sounds are readily elicited by tapping against the stomach walls and setting in motion the gastric contents. As a means of outlining the stomach the stroking method with the phonendoscope is a very convenient procedure. The stem of the instrument is placed over the stomach just to the left of the median line in the epigastric

region, midway between the ensiform cartilage and the umbilicus, and with the finger light strokes are made in different directions radiating toward the stem of the instrument. It is best to use only one ear piece in carrying out this method. As the finger passes over the border of the stomach a change in the sound is perceived. The points where this change takes place are marked and in this way the outline of the organ—at least of the cardiac extremity—is obtained.

*Palpation.*—Palpation is more applicable to the other abdominal organs than to the stomach. New growths are exceedingly rare and pyloric hypertrophy is not always sufficiently marked to become palpable, but it may at times be determined. Points of tenderness, may be elicited by palpation; and we must not lose sight of the fact that ulcer of the stomach may be present at an early period.

The interpretation of the *symptoms* referred to the stomach is an important point in diagnosing.

*Vomiting.*—Persistent vomiting from birth indicates either cardiac or pyloric obstruction. In the former deglutition sounds are absent, while in the latter the food is generally retained abnormally long before being rejected. At the same time dilatation of the stomach develops together with other signs of pyloric obstruction.

The natural tendency for infants to vomit must not be lost sight of. The cardiac sphincter is poorly developed and owing to the habit of gulping the food too rapidly or overfilling the stomach, vomiting is a common symptom. When the milk is too rich in fat it regurgitates shortly after nursing without being curdled. In indigestion the food is usually vomited an hour or more after nursing and it is curdled and sour. In acute gastritis there is fever; the food is promptly rejected and mucus is present in the vomit. In pyloric obstruction vomiting takes place after the stomach has become overfilled. The vomiting of intestinal obstruction is forceful and persistent; at first, gastric contents are rejected and later fecal matter appears. The vomiting of brain disease (*reflex*

*vomiting*) is projectile and unassociated with any gastric derangement. It is not easily differentiated from ordinary infantile vomiting. *Cyclic vomiting* is periodic; it occurs in older children and is due to auto-intoxication. The vomit often contains acetone and diacetic acid.

*Pain.*—Gastric pain in infants is usually spoken of as colic, although colic is perhaps more frequently intestinal than gastric. Gastric pain has been referred to above. Its true nature cannot be determined until the case has been investigated in every detail.

The final step comprises the **chemical examination** of the gastric contents.

The gastric contents are recovered by means of a soft rubber catheter into the free end of which a piece of glass tubing about three inches long should be inserted. For an infant three months old I use a number ten (English), from the third to the sixth months a number eleven, and from six months up a number twelve. It is well to slightly enlarge the eye of the catheter in order to permit the chyme to escape more readily. As soon as the tip of the catheter reaches the fundus of the stomach the chyme, as a rule, flows out freely if the stomach be full. Should there be difficulty in obtaining a specimen, the child may be bent forward and gentle pressure made over the pit of the stomach. This failing, it is better to withdraw the catheter and clean it in case it has become clogged with mucus or curds, then reintroduce and make another attempt. I do not approve of using suction, as I have seen it bring blood even when cautiously employed. By this means we obtain a specimen of the gastric contents for *inspection*, and can judge of the state of the digestion and whether mucus and blood be present. We also determine how long the food remains in the stomach. Ordinarily the infant's stomach is practically empty after one hour; in young infants it may be empty three-fourths of an hour after nursing, and under all conditions it should be empty after two hours in an infant one year old. In pronounced cases of

dilatation and atony the entire amount that was taken may be recovered after two hours.

The coagulated state of the milk indicates the presence of *rennin*. *Mucus* in small quantities may be normally present, but excessive amounts of thick tenacious mucus always stand for gastric catarrh. *Blood* not infrequently appears in the gastric contents, and, contrary to what may be expected, it rarely signifies ulceration. This is especially so of bright blood, which, in the majority of instances, originates from capillary oozing from the mucous membrane. In aphthous ulceration of the stomach the blood is usually dark in color.

The *odor* is also important to note. Butyric acid and acetone are detected by their odor. Fermentation may also be detected in cases fed on malt foods.

In order to estimate the amount of *free* and *combined hydrochloric acid* we must use a test-meal of barley water, as milk combines so energetically with HCl that no trace of free acid can be detected in the chyme. In some cases we must withdraw the test-meal at the end of three-quarters of an hour or we will find the stomach entirely empty.

The method of estimating the acidity which I personally follow is a simplification of the titration method. I have described it and my instrument in a previous article (*Hahn. Monthly*, May, 1903, A Study of the Gastric Contents in Infancy). The procedure is as follows: Pour the filtered stomach contents into the acidometer (Fig. 30) up to the line marked "S." Add a drop of dimethyl-amido-azo-benzol (if free hydrochloric acid has been detected previously by Boas' test) and then pour in, drop by drop, decinormal soda solution under gentle agitation of the tube. As soon as the red color is changed to orange, read off the amount of soda solution that was required and multiply by twenty. The product represents the amount of free HCl (*i. e.*, the number of c.c. of decinormal soda solution necessary to neutralize the free acid in one hundred c.c. of stomach contents). Now add a drop of phenolphthalein and continue adding soda solution until a pink color is ob-



tained; read off the number of c.c. required and multiply by twenty; this gives the combined acid on the same basis as above. The product of the sum represents the total acidity. The examination for free HCl and the calculation of the total acidity are data of the highest importance in gastrological work. Regarding the total acidity in infants I have formulated the following rule: Normal, thirty to forty; in indiges-

tion and mild grade catarrhal conditions it ranges between fifteen to twenty-five, while an acidity below ten indicates a grave condition, *i. e.*, either acute gastritis, chronic atrophic gastritis or marasmus. In the newborn—ten days old—I have found the acidity to be ten, pretty constantly. It gradually rises as the infant grows older.

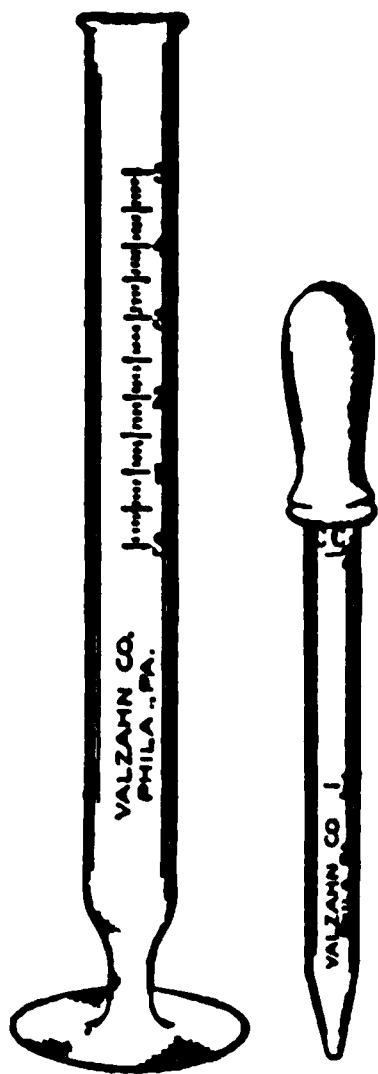


FIG. 30.—AUTHOR'S ACIDOMETER FOR ESTIMATING THE ACIDITY OF THE GASTRIC CONTENTS.

*Lactic Acid.*—It is claimed that lactic acid is found normally in the stomach in the early stages of digestion. Under these circumstances the amount is exceedingly small. Lactic acid that can be demonstrated in the gastric contents by Uffelmann's test is always abnormal. As hydrochloric acid inhibits lactic acid fermentation, the two practically never occur together. Of course, it is sometimes

possible to demonstrate a trace of lactic acid at the very close of digestion when a milk diet has been used and the stomach is almost empty, as under these circumstances the secretion of HCl has ceased, or it has all combined with the casein, and a trifling amount of lactic acid fermentation may take place. This is seen in infants with dilatation and atony. In cases of gastritis, gastric catarrh and marasmus, where hydrochloric acid is absent, lactic acid is invariably found when milk or a

food containing lactose has been administered. The invariable absence of hydrochloric acid in marasmus is a fact I have been able to prove conclusively after a thorough investigation of the subject. For a more detailed discussion I must refer to the article mentioned above.

Uffelmann's reagent consists of a weak, watery solution of neutral ferrous chlorid to which a few drops of a 5 per cent. solution carbolic acid have been added. This imparts to the reagent a steel color. Lactic acid changes the reagent to a canary-yellow color, while HCl only decolorizes it. Platt makes the test by simply diluting the ferrous chlorid with water to a point where only a trace of color remains; the same reaction is produced in this reagent by lactic acid.

*Ptyalin*.—In order to demonstrate the presence of ptyalin the infant should be slowly fed on an amylaceous liquid (barley water) and the stomach contents withdrawn after half an hour. It is then filtered and a few drops of a weak solution of iodine added. If only a blue color is obtained, ptyalin is absent; if, however, the solution assumes a pink or an orange color, starch digestion is taking place.

*Pepsin*.—I have repeatedly made the test for pepsin and have found it either absent or very feeble. It is done as follows: Place clean cut pieces of boiled white of egg into the gastric contents and acidulate with HCl if no free acid is present. The tube containing the test should be kept in warm water at 95 to 100° F. for several hours. If pepsin be present, the albumen gives evidence of being digested. The solution may also be tested for peptone (first boil to exclude albumen and then precipitate with picric acid)

*Absorption and Motility*.—The amount of food recovered at the end of one hour gives a clue to the motility of the stomach and its absorptive power. This method is more practical than the salol test. For testing gastric absorption a small amount of iodid of potash in solution may be poured into the stomach through a tube. The saliva is then tested with starch paper at intervals to note the time required to detect iodine in the same.



## ACUTE GASTRIC INDIGESTION; DYSPEPSIA.

An attack of indigestion in an infant or in a young child may induce beside the symptoms referable to the stomach others of quite an alarming character. If their true nature be not interpreted a serious error in diagnosis may result. Ordinarily, there is only the general discomfort, nausea and vomiting caused by the presence of undigested food in the stomach that may appear at any age, but under certain conditions, which no doubt depend upon bacterial activity in the gastric contents, the evidences of an *acute intoxication* are added. In such cases general convulsions frequently set in. The temperature may run high and remain so for several hours; periodic attacks of this nature are often mistaken for malaria.

Vomiting as a rule gives speedy relief, but even after free emesis fever may develop and the evidences of an acute gastritis set in, or if the stomach has not been completely emptied, intestinal disturbances may be added.

**Etiology.**—An attack of indigestion during early infancy is easily explained. The stomach has feeble digestive powers, and is very intolerant to any form of irritation. If food be taken in larger quantity or in more concentrated form than the stomach can manage, it will not be digested; irregularity in feeding, especially too frequent feeding, is also a prolific cause of indigestion. If the food contains micro-organisms it will decompose shortly after entering the stomach, with the formation of gases, acids, and in extreme cases toxines.

Saliva is not secreted in appreciable quantity during early infancy, nor is its power to convert starch into sugar developed until after the third month; for this reason amylaceous food is very prone to induce indigestion. Saccharine food, owing to its liability to fermentation, generally brings on an attack of flatulent dyspepsia when administered in excess.

In older children the same exciting causes active in adults are frequently found. Irregularity in eating is a most prolific cause of indigestion in children, and as they are very likely to

overeate, they frequently suffer with such attacks. Chilling the stomach with ice-water and ice-cream, and indulging in indigestible substances like nuts, fruit-cake, cheese, etc., are very prone to induce the painful form of indigestion, while fats and pastry rather induce nausea and vomiting, and candies and cakes the flatulent type.

**Symptomatology.**—In infants the first symptoms that will attract our attention are restlessness, crying and vomiting. The vomited matter consists of curds, undigested or partially digested food, as the case may be, and is usually mixed with serous fluid and acid mucus. The acidity is mainly due to the presence of lactic acid, as free hydrochloric acid is rare during infancy.

Should the stomach not empty itself completely, severe constitutional symptoms will occur from the absorption of albumoses and products of decomposition. The child develops a high fever, becomes apathetic and prostrated; the tongue becomes coated, the epigastrium bloated, and diarrhœa supervenes. This condition is frequently preceded by general convulsions.

Older children are usually feverish, complain of headache, nausea, and more or less gastric pain, while the advent of free emesis is followed by decided relief. The food sometimes remains for hours in the stomach in a partially digested, decomposing condition.

A mild attack of indigestion may subside spontaneously after the stomach has been relieved of its contents either by vomiting or fasting; but a more pronounced attack may be the exciting cause for an acute gastritis that may require considerable after-treatment.

**Treatment.**—Prophylaxis consists in feeding the infant on a pasteurized modified milk containing the proper percentage of proteids, fat, and lactose according to the requirements and digestive ability of the case, given in the proper quantity and at regular intervals. If these conditions are carried out the infant will rarely suffer from indigestion (see Chap. IV, "Infant Feeding").

Starchy foods, excepting the cereal-water diluents, should never be given to a child before the eruption of the teeth has taken place, and then only cautiously until the molars have made their appearance.

The first step in treating a case of acute indigestion is to empty the stomach. Sometimes it is but necessary to allow the infant to take several draughts of warm water, but the most satisfactory method is *lavage*. (See p. 24.) The process should be continued until the water comes out perfectly clear, and all traces of food and mucus have been removed from the stomach. In acute conditions one washing usually suffices.

The stomach should now receive a rest for several hours and feeding be resumed cautiously, beginning with a cereal water. In the course of twelve to twenty-four hours if the symptoms have subsided milk may be cautiously added, beginning with low percentages of fat and proteids. Heubner uses a tablespoonful of rice-meal shaken up with cold water, and subsequently boiled with one pint of water for a quarter of an hour and sweetened with a few teaspoonfuls of milk sugar. According to Rotch, a modified milk containing less than 5 per cent. lactose, about 3 per cent. fat, and in extreme cases as low as 0.45 per cent. proteids, will have to be used until the digestive function of the stomach becomes normal. Naturally, this will not apply to many cases, and would be unnecessary starvation in most instances. Personally I give less fat and more proteids than represented in the above formula.

In older children it is not so easy to employ lavage, and a simpler method, such as instructing them to drink warm water and then inducing vomiting reflexly by irritation of the fauces, is often more satisfactory. A tongue depressor passed far back over the base of the tongue is a good method of inducing vomiting. At the same time the throat can be fully inspected—a most important procedure in all acute illnesses associated with gastric symptoms. It is seldom, however, necessary to employ any artificial means, as children proverbially vomit on the slightest provocation.

Remedies are seldom necessary in infants after the stomach has been emptied and the diet carefully regulated. However, there are cases in which attacks of indigestion will recur despite the greatest care in these respects. Here a remedy is necessary to correct the underlying disturbance. In older children, who can relate their symptoms, we are often capable of averting an attack by an early prescription.

*Abies nigra*.—Sensation of a hard-boiled egg in stomach.

*Ethusa*.—Vomiting of large curds of milk, followed by great exhaustion.

*Antimon. crud.*—Tongue white, heavily coated; great nausea; results of overeating.

*Arsenicum*.—After chilling the stomach with ice-cream or ice-water; nausea, prostration.

*Bell.*—Throbbing headache; strawberry tongue; convulsions. It is the best remedy for the febrile cases with irritation of the nervous system, *i. e.*, *gastric toxæmia*.

*Bry.*—During summer and sultry weather; anorexia, thirst, mouth dry, distress and pain in stomach, as of a load.

*Ipecac.*—Nausea and vomiting, tongue usually clean; stomach feels relaxed.

*Nux vom.*—Tongue coated at base; bitter taste; painful pressure in stomach; great desire to vomit—urges to do so; ineffectual urging to stool; headache and vertigo; quarrelsome disposition; face hot; chilly feeling.

*Pulsatilla*.—Tongue coated and dry; mouth feels pasty and contains thick saliva; no thirst or appetite; water tastes bitter; nausea and faintness; languor and feverishness; diarrhœa. After rich food, pastry and cold food (*Ars.*). Often relieves when *Arsenicum* does not act.

#### CHRONIC GASTRIC INDIGESTION; NERVOUS DYSPEPSIA.

A chronic condition of disordered digestion is seldom found without a definite pathological process involving the mucous membrane of the stomach, although there is sufficient clinical evidence that there may be a purely neurotic type of

indigestion. Hyperacidity and increased secretion of the gastric juice; diminished secretion of gastric juice; sensory disturbances, and insufficiency of motor power of the stomach have all been observed as purely functional phenomena, although in the majority of cases a mild grade of gastritis accompanies these conditions, for which reason differentiation between the two is often impossible, the preponderance of the symptoms of the one condition over the other deciding the diagnosis. Hyperchlorrhœdia, the commonest form of nervous dyspepsia in adults, is only occasionally encountered in children. The majority of cases of indigestion still belong, as in infancy, under the category, *improper feeding*.

Anæmia; neurasthenia; lithemia; adenoid vegetations; decayed teeth—are prominent factors in the etiology of dyspepsia. No single cause, however, should be looked upon as final, but the child's constitution, hygienic surroundings, habits and food should be thoroughly looked into before a case can be intelligently and successfully treated.

**Symptomatology.**—The motor power of the stomach is usually deficient, which allows the food to remain an undue length of time in the stomach, thus favoring fermentation and the production of gas, together with lactic, acetic, butyric and other acids. Regarding the anomalies in secretion, Leube found that there is either a diminution or excess in the acidity of the gastric juice, although dyspeptic symptoms are often encountered where the secretion is both normal in quantity and quality, and where the food could not be found remaining abnormally long in the stomach (neurotics).

The subjective symptoms complained of are distress and uneasiness after eating; sometimes malaise, headache, vertigo, and restless sleep may be observed. The appetite usually becomes impaired and capricious and the belching of gas or the eructation of food or a sour liquid is common. Intestinal indigestion is so frequently associated, and the symptoms of the one so gradually merge into those of the other, that a sharp line of distinction is impossible.

*Hyperchlorrhya* is a neurosis in which an excessive secretion of hydrochloric acid takes place. It is encountered in neurasthenia, especially in those with gouty antecedents. The symptoms are burning and distress in the epigastrium; sour eructations and often nausea and vomiting. The paroxysms come on several hours after eating and the symptoms are relieved by eating. Often there is continuous gnawing in the stomach and craving for food.

In *infantile indigestion* there is almost always vomiting, and if the contents of the stomach be examined, stringy mucus, lactic acid and sometimes butyric acid will be found. Flatulency is also prominent, the intestines as well as the stomach sharing in the production of gas. These cases are usually described as colicky babies, for they rarely seem to be without pain. The treatment will be taken up in connection with "Chronic Gastritis," p. 161.

#### ACUTE GASTRITIS.

Acute gastritis may be encountered in the form of a catarrhal, follicular or pseudo-membranous inflammation of the mucous membrane of the stomach.

**Etiology.**—Although gastritis in one of its forms is frequently met with unexpectedly during an autopsy, we are, on the other hand, often disappointed by finding no definite lesions where the condition had been thought to exist during life. Causes which seem to excite gastritis in one individual produce nothing more than functional indigestion in another case. For this reason the etiological factors for indigestion must be looked upon as capable of also producing gastritis under certain circumstances, such as malnutrition, scrofula and rickets, unsanitary surroundings, especially dearth of pure air and sunshine and insufficient clothing. Among other causes as producing gastritis, such indefinite terms as "improper food or feeding" and "exaggerated form of indigestion" are mentioned. The majority of cases no doubt result from bacterial toxins or decomposed food.



Micro-organisms do not propagate in the stomach as well as in the intestinal tract, for which reason gastritis is less common than enteritis, and when present it is usually accompanied by enteritis (gastro-enteritis, summer-complaint); but it is to be remembered that the most prolific cause of gastritis in infants is the absorption of toxines, which have developed already in the food before entering the stomach. It may stop just short of, or develop into, cholera infantum.

Any irritant may induce gastritis when taken in sufficient quantity; many drugs come under this heading. Food given too hot has induced it; also ice-cold foods and drinks.

Direct infection has occurred during diphtheria and other infectious diseases, resulting in the membranous variety.

*Corrosive gastritis* is usually the result of the accidental introduction of an acid or caustic into the stomach, and belongs to the domain of toxicology.

The acute infectious diseases, such as scarlatina, pneumonia, typhoid fever and septic conditions, especially when the intestinal tract is involved, are often accompanied by gastritis. Follicular gastritis is undoubtedly of infectious origin. It is most frequently encountered in the newborn and in early infancy.

**Pathology.**—The *catarrhal variety* is the one most frequently met with, and presents the usual signs of catarrhal inflammations elsewhere. The mucous membrane is hyperæmic and swollen; the sub-mucosa more or less infiltrated with round cells and distended with serous exudate.

Here and there injected areas, small hæmorrhages and superficial erosions of the mucous membrane will be found. The mucous membrane is usually most markedly affected at the pyloric end and along the greater curvature. The contents of the stomach consist of undigested and partially digested food and mucus, or it may contain only thick tenacious mucus and serous fluid, with an admixture of brownish, decolorized blood. If the stomach is distended it usually contains offensive gas.

The *follicular variety* is rare, is usually associated with catarrhal gastritis, and has no specific etiology. It is characterized by swelling of the solitary lymph follicles of the stomach with secondary softening and necrosis, resulting in small, scattered ulcers. They are seldom large enough to attract attention, and require the microscope for verification, but in some cases there is sufficient ulceration to induce hæmorrhage and other symptoms of ulcer. In *melena neonatorum* this condition is found associated with similar ulcers in the gut.

The *membranous variety* of gastritis is also of rare occurrence, and is always secondary to some infectious disease. It has been observed after diphtheria, pseudo-diphtheria, scarlatina, variola, typhoid fever, pyæmia, and in conjunction with membranous colitis. The membrane is grayish-green in color, and is composed of fibrin, epithelium, bacteria and *débris*. The mucous membrane is thickened and eroded beneath the pseudo-membrane. Pieces of the membrane are at times found in the vomited matter.

**Symptomatology.**—When any of the pathological changes mentioned above have developed to an appreciable degree, the symptoms of an *acute febrile gastritis* are the invariable result. The *afebrile* variety of gastritis is not so severe in its course, and must be considered a very mild grade of gastritis, or in some instances a subacute form of the disease; indeed, it is often impossible to draw a sharp line of distinction between afebrile gastritis and indigestion (functional). Rotch believes that the majority of cases of so-called gastritis catarrhalis are nothing more than functional disorders.

The early symptoms are those of acute indigestion, namely, coated tongue, nausea and vomiting, pain, prostration, feverishness. Vomiting is the most prominent symptom, and is usually stubborn.

Booker has shown that the food may lie from four to five hours in the stomach in these conditions, and for this reason the ejecta usually consist of undigested food, beside sour mucus. If the vomiting persists for a long time bile eventu-



ally appears, and fermentation, with the production of gases and consequent flatulent distension of the epigastrium, takes place. I have often noticed when children are fed on artificial foods containing malt, the stomach contents will ferment and the odor be strongly suggestive of stale beer. Dilatation of the stomach is a natural result of this abnormal condition.

In my series of sixty reported gastric analyses in children (*Hahnemannian Monthly*, May, 1903) there were six cases of primary acute gastritis. In all, hydrochloric acid was absent from the stomach contents, while mucus and in two blood and mucus was present. When milk had been taken as a food, lactic acid was present. One case in which considerable blood appeared in the ejecta and in which ulceration was suspected showed no evidence of the same at the autopsy.

Older children complain of headache, dizziness and nausea, while the infant makes its discomfort known by fretfulness, crying and great restlessness; the pulse is small and rapid, and the extremities cold. If the gastric symptoms do not disappear within two days diarrhœa usually sets in.

The *febrile* variety is more characteristic in its course, and points to decided involvement of the gastric mucous membrane. It is sudden in onset, beginning with high fever ( $103^{\circ}$  to  $104^{\circ}$ ), vomiting and prostration. The tongue is heavily coated and may show the imprints of the teeth; the breath is offensive and vomiting persistent, even drinks being ejected as soon as they reach the stomach. In the beginning food and mucus constitute the vomited matter; later, bile may appear. The mucus is frothy and sour, often containing blood.

Epigastric tenderness is marked; the child is exceedingly restless in the beginning from the pain and thirst, later becoming prostrated; the circulation weakens, the extremities become cold, and a clammy sweat breaks out on the forehead.

Thirst is a prominent symptom, but appetite for food or tolerance for the same are characteristically absent.

The fever generally falls after the second day, and ranges

between  $100^{\circ}$  and  $101^{\circ}$  for several days, until at the end of five days or a week it has regained the normal standard. Intestinal symptoms usually supervene in infants, and may prolong the course of the disease. Hydroa frequently develop on the lips.

In older children the temperature does not range so high, nor is there as much prostration.

The *prognosis* is favorable excepting in debilitated or cachectic infants. The *membranous variety* can only be diagnosed when pieces of membrane are vomited.

**Diagnosis.**—From *simple indigestion* gastritis is not easily differentiated in the beginning, but the presence of abundant mucus in the vomited matter, with at times blood; absence of hydrochloric acid in the gastric contents; tenderness over the stomach; the longer duration of the attack, symptoms continuing even after the stomach has been emptied and the diet regulated, beside the elevation of temperature, must lead us to suspect gastritis and exclude functional indigestion.

The *febrile* variety is most likely to be confused with beginning *typhoid fever*; however, the absence of nose-bleed and typical step-like rise of temperature, beside the absence of typhoid roseola and enlarged spleen, and the history of some dietetic error and presence of herpes labialis, will differentiate the two affections.

The *subacute* form is most readily diagnosed by removing the contents of the stomach with the lavage apparatus, one to two hours after a test-meal of barley-water. The washings will contain particles of undigested casein if milk was previously taken; abundant mucous secretion; lactic and fatty acids, but no free HCl.

**Treatment.**—The *non-febrile* variety is to be treated in the same manner as indigestion, employing lavage once or twice daily, especially if there is much mucus and acid fermentation, and withholding all food for a period of six to twelve hours, as the condition of the child may suggest. Feeding should be resumed cautiously, beginning with one of the

preparations recommended under acute indigestion. Remedies are more necessary here than in simple indigestion. Older children should be put to bed, and likewise fed cautiously on well-diluted milk to which lime-water or seltzer can be added. Weak tea, beef- or mutton-broth, or one of the reliable proprietary foods are also permissible. Cracked ice is most useful for the thirst and dryness of the mouth, often controlling the vomiting. If it is not effectual in this respect, hot water may be sipped.

The *febrile* form demands even a more strict mode of treatment; here food is best withheld for twelve to twenty-four hours, and, if the child be feeble, rectal alimentation, alcohol sponge-baths and well-diluted brandy, must be resorted to. Cracked ice, hot water or albumin-water by the teaspoonful is all that should enter the stomach until the fever abates and the retching and pain cease. Lavage must be used with caution, although it is usually effectual. When vomiting becomes uncontrollable, the stomach should be put at absolute rest. High rectal enemata of normal saline solution are most useful to control thirst and prevent suppression of urine and aid in the elimination of toxins from the blood. They are especially valuable when cerebral symptoms are present.

**ACON.**—After exposure to cold; great thirst and restlessness; high fever; anguish.

**ANT. CRUD.**—Anorexia; tongue heavily coated, as if white-washed; after Christmas and Thanksgiving dinners; over-eating.

*Ant. tart.*—Persistent vomiting, with tendency to collapse.

*Apis.*—Epigastrium sensitive to touch; yellowish diarrhoea; scanty urine.

*Arnica.*—After overeating; belching of putrid gas, tasting like rotten eggs; head hot, extremities cold.

**ARSENICUM.**—After ice-water, ice-cream; great thirst, taking little at a time; vomits when rising; prostration marked; restlessness.

**BELL.**—Full, bounding pulse and high fever; coated tongue with prominent papillæ; thirsty, but drinking aggravates. Cerebral symptoms.

**BRY.**—Loss of appetite; great thirst for large quantities of water; sensation of a load in stomach.

*Ferrum phos.*—Inflammatory stomach-ache in children from chill, with diarrhœa.—(BOERICKE and DEWEY.)

*Cham.*—Vomiting of bile; cheeks flushed; fretful and irritable temperament.

**GELS.**—Fever, with drowsiness; soft pulse; nausea and dizziness.

**IPECAC.**—Constant nausea; tongue clean; after unripe fruit or sour things; also after rich food (*Puls.* has coated tongue and bitter taste).

*Iris.*—Great burning in mouth, œsophagus and stomach; vomiting and diarrhœa, with great prostration; the vomited matter is very acid; headache over eyes.

**MERC. DULCIS 2x.**—"Will cure a majority of all cases in children."—(HALE.) This remedy I consider the most useful for controlling the vomiting and cleaning the tongue.

**NUX VOM.**—Nausea, with great desire to vomit; vertigo; frontal headache; irritable disposition; bowels constipated; after the use of coffee, condiments or irritating medicine, quack nostrums, etc.

*Podophyllum.*—Ejected matter very sour; expulsive effort of stomach so violent that it causes the child to cry out with pain; vomiting of bile tinged with blood; diarrhœa.

**PULSAT.**—Tongue coated white, or yellowish and dry; no appetite; loss of smell and taste; no desire to drink, while the mouth is dry and contains sticky saliva; water tastes bitter; nausea several hours after eating; diarrhœa; vertigo and chilliness; after rich food, pies and pastry.

*Sanguinaria.*—Burning in throat and stomach; sick headache; tongue and lips red and dry; nausea and vomiting.

*Sepia.*—Epigastrium sensitive; urine profuse and clear, later scanty, with red deposit; tongue coated, without luster;

herpetic eruption on tip and along its edges ; “ especially in children after taking cold when the weather changes.”—(C. G. R.)

VERATR. ALB.—Persistent vomiting ; cold sweat on forehead ; hippocratic countenance ; coldness of extremities ; hæmatemesis ; nausea, worse from rising or moving ; purging.

#### CHRONIC GASTRITIS—CHRONIC GASTRIC CATARRH.

Chronic gastritis presents in its milder form many of the symptoms of simple functional indigestion, and as the latter condition is frequently accompanied by a low grade of gastritis, the two conditions are by some authors described under the same heading. There are, however, definite pathological changes which affect the mucous membrane primarily, and the muscular coats secondarily, in true cases of chronic gastritis, making it a separate clinical condition.

**Etiology.**—In infants the most frequent cause of chronic gastritis is improper feeding, both as to intervals in time of feeding, and quantity and quality of the food. Food of an indigestible nature is often given continuously for a long period of time (such as amylaceous preparations or milk of an abnormally high proteid percentage) or unsterilized nursing-mixtures are administered, whereby the stomach becomes irritated from the fermentation going on during digestion.

In older children repeated attacks of acute gastritis are likely to assume a chronic nature, but here, also, dietetic errors are the most frequent exciting cause of the disorder. It is rare, however, for a healthy child to become a victim of chronic gastritis ; as a rule there is some predisposing constitutional disease. In adults, the use of liquors, strong condiments and spices, rich, indigestible food, and late suppers, together with the cares of business and the exhausting struggle for existence, often induces a pronounced gastritis in a person otherwise sound in body ; but healthy children, not being subjected to this mode of living, naturally are exempt. Where, however, we have tuberculosis, rickets, syphilis, organic

heart disease and nephritis, causes which would ordinarily remain inactive, or at the most produce simply functional disturbances, are often sufficient to bring about definite pathological changes in the stomach. In valvular disease of the heart and cirrhosis of the liver the passive congestion of the stomach eventually results in gastritis.

**Pathology.**—The mucous membrane is of a pale gray color, covered with tenacious mucus, and may be thickened and show injected areas. These changes are most pronounced at the pyloric end of the stomach. Microscopically, the epithelium of the tubules is found to exhibit degenerative changes. In the submucosa there is round-cell infiltration, which may invade the glandular structure, inducing atrophy of the tubules. The stomach is usually dilated, and in extreme cases the mucous membrane is atrophied and smooth, while the submucous and muscular layer are much increased in thickness.

**Symptomatology.**—The cardinal symptoms are increased production of mucus, vomiting, indigestion and malnutrition. In infants there is always more or less involvement of the small intestines. Naturally in such cases malassimilation becomes the most prominent symptom and the infant succumbs to marasmus. If the stomach be irrigated abundant mucus appears in the washings.

Vomiting usually occurs some time after eating, the food being but partially digested. In older children it may occur in the morning before breakfast, as in adults, and consists of thick, glairy mucus.

The process of digestion is slow, both on account of the presence of mucus and the deficiency in hydrochloric acid, as well as the deficient motor power of the stomach. For this reason we will find food in the stomach four or five hours after eating. This invites fermentative changes with the formation of gases and such acids as acetic, lactic and butyric, causing eructations and heartburn. Secondly, the production of these acids gives rise to intestinal irritation, as the intes-



tinal juices are not adequate for the neutralization of this excessive acidity, and an acid diarrhœa may result; or, if the acids be absorbed into the general system, all the evils of the acid dyscrasia will be encountered. Constipation is also frequently associated.

Parrot divides the condition above described—namely, where gastric catarrh exists together with intestinal disturbance—into three stages, giving it the name *athrepsia* in severe cases. The first stage marks the advent of the gastric catarrh, with its symptoms of vomiting, colic, flatulent distention of the abdomen and diarrhœa. Next, progressive wasting makes itself manifest, followed by the stage of exhaustion. The child becomes dull and apathetic, the cry feeble, and death ensues, usually preceded by convulsions.

Ewald ("*Diseases of the Stomach*") recognizes three clinical varieties of chronic gastritis, the distinction being mainly based upon the severity of the disease and the stage to which it has progressed. Thus, the first variety is described as *simple chronic gastritis*, in which the fasting stomach contains a small amount of thin, yellowish mucus, the hydrochloric acid is diminished, and lactic and fatty acids are usually present.

*Chronic mucous gastritis* is characterized by the presence of a large amount of mucus and absence of hydrochloric acid.

*Atrophy of the gastric mucous membrane* is the final stage of both forms, and here there is neither mucus nor gastric juice to be found.

Other symptoms usually found in chronic gastritis are coated tongue, bad taste in the mouth, distress after eating, variable appetite, distended abdomen, constipation. The urine contains urates in excess, and often phosphates.

As atrophy of the mucous membrane sets in, a condition of true stomachic indigestion is established. Nutrition becomes much impaired and a high grade of anæmia develops, although the intestines greatly compensate for the disabled stomach in many instances. In pernicious anæmia complete atrophy of the gastric mucosa is frequently encountered.

**Prognosis.**—Unless associated with a serious constitutional dyscrasia or some incurable form of heart or kidney disease, chronic gastritis is curable in its early stages, and generally shows prompt improvement under the proper form of treatment. Nothing can be done for the stage of atrophy, but it is rarely met with in children, as they either recover or succumb from athrepsia before the stage is reached. In very young and delicate children the prognosis is unfavorable unless the process can immediately be checked, as they lack the vitality necessary to rally from the resulting exhaustion.

**Diagnosis.**—From the functional form of *indigestion* chronic gastritis is readily distinguished by examining the contents of the stomach. The presence of an abundance of mucus and undigested food four to five hours after eating and decreased hydrochloric acid secretion, together with coated tongue, vomiting of mucus and possibly the association of some constitutional disease or heart and kidney trouble, must at once exclude nervous dyspepsia.

The different varieties of *chronic gastritis* are differentiated by chemical examination of the stomach contents, as described under the symptomatology.

*Tuberculous meningitis* may be suspected from the presence of vomiting and wasting; here, however, there is fever, irregular pulse, bulging of the anterior fontanelles, and definite nervous symptoms.

**Treatment.**—The treatment of chronic indigestion and chronic gastritis is practically carried out on the same lines. Prophylaxis is a most important factor. Pure air, pure water and a perfect condition of the skin are a necessity for all cases predisposed to these affections. The diet must be carefully supervised. In infants, the time for feeding and quantity for each feed must be carefully determined; so also the sanitary condition of the food—namely, it must be uncontaminated by bacteria and be absolutely fresh. This applies not only to milk but includes especially fruits in the summer months.

In infants milk should be withheld entirely until vomiting



and the excessive secretion of mucus has been controlled, feeding the child during this time upon broths with a little rice or barley, thoroughly boiled. Albumen-water and raw meat juices are also to be fed at this stage. When milk is resumed it should be given in low percentages of fat and proteids; the relation of these elements most likely to agree is two of fat to one of proteids. As there is proteid indigestion in these cases, the percentage must be cut down until the digestive powers become improved.

In older children sugar and starch must be indulged in sparingly, as they favor fermentation. Rich food and fried dishes are to be avoided. Early in the treatment it is often wise to restrict the diet to milk; later, soft-boiled eggs, chops, toast, succulent vegetables, fruit and some of the cereals, especially rice; may be added.

*Lavage* is particularly serviceable, and as it can so readily be carried out in young children, it is one of the most important therapeutic measures at our command. Indeed, many cases show immediate improvement after the first few washings, more particularly in the subacute form, and rapidly recover when the indicated remedy and the proper diet are at the same time administered.

In many instances the condition improves under constitutional treatment more promptly than by simply taking the stomach into consideration, as it is so frequently only the outcome of a general disturbance.

The remedies recommended for chronic indigestion are numerous, but there are a few which have stood the test of time, and which should therefore be considered first in connection with the disease.

*Pepsin*, the saccharated preparation, is a very useful palliative.

*Nux vom.* is frequently indicated, especially if the patient has been dosed with harmful remedies. We must not forget, however, that *Strychnine* is frequently employed in these cases, and here it is better to begin with *Pulsatilla*, especially

if there is the pasty, heavily-coated tongue ; mouth dry, containing a little thick mucus ; no thirst ; distress or vomiting occurring an hour or more after eating ; symptoms developing after rich foods and pastry ; belching which gives relief ; in younger children usually vomiting of undigested food containing mucus, and diarrhœa.

*Atropin sulph.*, 3x trit., one to two tablets after meals, acts most satisfactorily in hyperchlorrhydria to check the excessive secretion of hydrochloric acid. Its action is purely palliative, however, and constitutional treatment, especially in conjunction with out of door exercise and sponge baths, is necessary to eradicate this neurosis. *Calc. carb.* is to be thought of as a curative remedy.

*Anacardium* is indicated in hyperacidity and gastralgia on the symptoms of pain and distress in the stomach relieved by eating.

*Kreosotum* is useful where there is much fermentation and vomiting of thick, glairy mucus.

*Hydrastis* is an excellent remedy in gastro-intestinal catarrh with constipation ; coated tongue ; loss of appetite ; gone feeling in stomach.

*Carbo veg.*, *Lycop.* and *China* are indicated especially by the presence of flatulence. *Carbo veg.* has belching of foul gas, with coated tongue and general venous stasis of the abdominal viscera ; *Lycop.* has flatus passed downwards, together with dark urine which stains the diapers or causes the child to cry during micturition, from its irritating quality. In *China* there is usually colic, induced by the gas ; also eructations without relief ; also diarrhœa, anæmia, and emaciation. Other remedies which may prove useful are :

*Arg. nitr.*—Flatulency and vomiting of quantities of ropy mucus, especially in the morning.

*Arsen.*—After chilling the stomach with ice-water or ice-cream. Morning vomiting. Relief from hot food ; drinks little and often.

*Bry.*—Loss of appetite, coated tongue ; thirst for large quantities of water at a time ; constipation.

*Calc. carb.*.—Scrofulous diathesis; desire for eggs; stools light and clay-colored. Acid dyspepsia in tuberculous subjects, the “pre-tuberculous stage” (Hughes).

*Euonymin.*.—Bilious type; tongue yellow, breath offensive; slow pulse.—(HALE.)

*Hydrastis.*.—Large, flabby tongue; catarrhal symptoms predominate; obstinate constipation.

*Ipecac.*.—After rich food and pastry; tongue usually clean; persistent vomiting.

*Phosphorus.*.—Regurgitation of food; vomiting of water as soon as it becomes warm in stomach.

*Sulphur.*.—Skin dry and harsh, old appearance of child; aversion to being washed; faintness and hunger in forenoon; bright redness of the lips; tongue furred in morning, wearing off during day; child never seems satisfied, constant craving for food; epigastrium sensitive to pressure, which causes eructations; eczema.

#### CYCLIC, OR PERIODIC VOMITING.

In recent years attention has been called to the periodic occurrence of attacks of vomiting in children, not the result of indiscretions in diet with consequent acute indigestion, but rather due to a toxæmia of intestinal origin (auto-intoxication). These children belong to the clinical type described as “lithæmic,” but uric acid is not the cause of the attacks. While uric acid may be discovered in increased amounts in the urine prior to the attack and appear diminished or insufficiently excreted during the attack, still this constituent of the urine must be looked upon rather as an index of the auto-intoxication than as the cause of the same. Rachford (*Archives of Pediatrics*, 1897) offers the most rational explanation of the cause of the attacks, namely, the formation in the system of alloxuric bodies of the xanthin group in sufficient amount to produce the symptoms noted. This group contains paraxanthin and heteroxanthin which are both toxic.

Other etiological factors are heredity and a neurotic tem-

perament. Norton (*Hahnemannian Monthly*, March, 1903) found these factors well marked in his three reported cases. Rachford, Larned, Holt and others express similar views.

**Symptomatology.** — Prodromes are not marked. There may be malaise, loss of appetite, a furred tongue and sourish breath, or the attack may be ushered in by vomiting. This is usually forcible, sometimes uncontrollable and the child may die of exhaustion (Marcy). In severe cases, as exhaustion sets in vomiting takes place without much effort and the vomitus consists of a thin fluid tinged with blood.

At first the gastric contents are ejected and when the stomach has emptied itself retching sets in with the vomiting of a thin fluid containing bile and a little mucus and blood. Blood, however, is not necessarily present. Food and drink are not tolerated. Hydrochloric acid soon disappears from the gastric fluid and acetone and diacetic acid can be demonstrated in some cases (Edsall). These elements are also found in the urine pretty constantly.

The bowels are constipated and the stools are lighter in color than normal, indicating a decrease in bile. Diarrhœa has been observed, but purging does not materially influence the vomiting. The abdomen is retracted, but there is no abdominal pain.

Fever is usually present. Ordinarily it is not high, but I have seen it reach 103° F. Anorexia is pronounced and there is intense thirst. The pulse is rapid and may become very weak, while the respiration is similarly affected. Convulsions have been observed.

Pruritus and urticaria may be associated with the attack.

The urine usually shows decided changes. It may be loaded with amorphous urates and uric acid, although at the height of the attack it generally becomes profuse and pale in color and of low specific gravity. A trace of albumen and granular casts are present in most instances. Acetone and diacetic acid are pretty constantly present (Morse; Edsall). One is more likely to find these products just preceding or in

the early stages of an attack (Pierson). Indican is present in increased amount, but disappears during the attack (Marcy).

The duration is from two to four days. Convalescence is usually rapid, although in some of Snow's cases it was protracted. There seems to be no distinct periodicity as to the recurrence of attacks. Einhorn, however, favors this view.

The *prognosis* is favorable in the majority of cases, but when the child is frail and the intoxication is pronounced it becomes grave. Marcy has reported two fatal cases.

**Treatment** — This must be chiefly prophylactic. The child's general condition is to be improved by fresh air, exercise and careful regulation of the diet. Meat should be given sparingly to these children, but there is no doubt that an excess of starchy food, notably potatoes and oatmeal, tends to provoke attacks. Fruit, fats and milk may be given freely and the regular drinking of water is to be insisted upon. *Nux vomica* and *Lycopodium* are usually indicated as there is more or less intestinal indigestion.

During the attack it is best to stop the administration of any food, giving water freely. In uncontrollable vomiting I make use of lavage. If the child be much prostrated this may be performed in the recumbent position. The bowels should be emptied by a glycerin enema (one ounce to six ounces of water), and this procedure followed by high colon injections of warm normal saline solution twice daily. By this means the distressing thirst is alleviated and the elimination of toxins hastened.

Pierson claims to have obtained beneficial results from the administration of *Sodium bicarbonate*. Its action is supposedly to neutralize the urine and aid in the diminution and neutralization of the uric acid, diacetic acid and acetone.

The most satisfactory remedy in my experience for controlling the vomiting and nausea is *Merc. dulcis*, 2x trit., two grains every two to three hours. Norton recommends *Cuprum ars.* and *Iris*.

## GASTRALGIA.

The purely neuralgic type of gastric pain, or gastralgia, is seldom met with in the very young, and, if so, cannot be diagnosed for obvious reasons. The dyspepsias of infants are generally associated with pain, especially when there is much flatulence, and, naturally, gastritis is accompanied by gastric pain; but in older children gastralgia without any objective disturbance can occur, as in the case of adults.

**Etiology.**—A neuralgia of the stomach may result from exposure to cold and wet, drinking ice-water or eating very cold food, malarial infection, and other causes likely to induce neuralgic pains elsewhere. Irritation of terminal filaments of the pneumogastric will induce the identical condition, and so hyperacidity of the gastric juice is a frequent cause of these attacks. Certain articles of food may induce it without producing actual indigestion in some individuals, and the use of lemon-juice, vinegar and other acids often brings on severe pain. Predisposing causes are anæmia, neurasthenia, rickets and tuberculosis; in some instances fright or anger seems to have precipitated an attack.

**Symptomatology.**—The pains are paroxysmal, coming at intervals, between which the patient is entirely free from any discomfort. The paroxysm may have such prodromal symptoms as yawning, pressure in the stomach and coldness of the extremities, or it may come on suddenly as a violent cramp, or as a pressing, burning or gnawing pain, so violent at times as to result in collapse. A characteristic feature of the pain seems to be its radiation from the spine and the tendency to reflect up into the region of the heart, often closely simulating angina pectoris. Sometimes the pain is temporarily relieved by eating. Firm pressure also may give relief.

**Diagnosis.**—Gastralgia is to be distinguished from several important conditions, notably *gastric ulcer*. This is rare in children; the pain is more constant; it is aggravated by eating; tenderness is also constantly present, and may be defin-



itely located at one spot. The vomiting of blood usually occurs, verifying the diagnosis.

In *indigestion* and *gastritis* there is the history of some error in diet or other cause for the trouble, together with vomiting and relief of symptoms thereby in the former, and continued fever, anorexia, vomiting of mucus and coated tongue in the latter.

Other conditions in which pain is referred to as being located in the epigastric region are *vertebral caries* in the dorsal region and *diaphragmatic pleurisy*, which must be differentiated by their own peculiar symptoms. In *pneumonia* and *pericarditis*—no doubt through the involvement of the diaphragm—epigastric pain is also frequently complained of. *Gall-stone colic* may also occur in children. Here the examination of the stools, the condition of the pulse, tenderness in the gall-bladder region and the subsequent symptoms will readily exclude simple gastralgia.

**Treatment.**—During a paroxysm the patient must be put to bed, although, as a rule, rest is impossible from the agonizing pain. Food should be withheld, and hot fomentations applied over the epigastrium. When this does not help, an ice-bag may be tried. Hot water internally, with a little brandy or gin added, often gives material relief. If the pain is uncontrollable a grain of *Acetanilid* with three grains of *Sodium bicarbonate* may be administered.

After an attack the patient's habits must be regulated and a hygienic mode of living carried out. Irregularity in eating must be corrected, also the excessive use of starchy and saccharine food. The diet should be highly nutritious and easily digested, especially in the neurotic class of patients. They should partake of milk, eggs, young meats, succulent vegetables, bread and butter and stewed fruit liberally. Sufficient exercise and fresh air are of great importance. Cod-liver oil may be necessary in the scrofulous or rachitic.

**ARSEN.**—The pains are usually of a burning character, or purely neuralgic, resulting from the abuse of cold drinks or traceable to anæmia or neurasthenia.

CUPRUM ARSENICOSUM has proven clinically perhaps the best routine remedy in gastralgia.

BELL.—Cramp-like pain extending into spine, relieved by bending backward (opposite to *Colocynthis*). Face flushed; thirsty, but drinking aggravates.

*Bismuth*.—Intense pressure on one spot; relief from bending backward.—(BELL.)

*Bryon*.—Pressure in stomach as of a heavy load. Relief from rest.

CALC. PHOS.—As a constitutional remedy between attacks. When anæmia is well marked, FERRUM PHOS. is preferable.

*Cham*.—Tossing about in agony; unmanageable; after anger or vexation.

COLOCYNTH.—Cutting pains concentrating in epigastrium, relieved by firm pressure and bending double (comp. *Bell*.).

*Ignatia*.—Hungry gnawing in stomach; faintness in epigastrium. Neurasthenia.

*Lycop*.—Hungry feeling, but sudden repletion after eating a few mouthfuls. Lithæmia; constipation; flatulence.

NUX VOM.—Clawing pain in stomach, extending into chest or downwards, producing retraction of anus; ineffectual urging to stool; the pains are relieved by rubbing the stomach, belching and vomiting; worse after eating, although there may be craving for food. Useful both during and between the attacks.

*Petrol*.—The pains are ameliorated by constantly eating something (*Anac.*, *Chelidonium*).

#### MALFORMATIONS AND MALPOSITIONS.

*Atresia* or *stenosis* of either the cardiac or pyloric end of the stomach in infancy is congenital; later in life it may develop as the result of an inflammatory process either in the stomach or adjacent viscera, or, what is more likely to be the case, after the accidental swallowing of some caustic or corrosive poison, or from a burn. In the latter instances the stenosis, as a rule, involves the œsophagus and cardiac orifice of the stomach.



The congenital form terminates fatally within a few days, while the acquired form may be relieved by surgical interference. Persistent vomiting is the only positive symptom. Locating the obstruction with the œsophageal bougie confirms the diagnosis.

*Spasmodic stenosis* of the pylorus may occur with every symptom of congenital stenosis, with this exception, that the obstruction is not permanent and the case recovers. It may, however, result in permanent stenoses by setting up an hypertrophy of the tissues about the pylorus. The condition is described below (see "Dilatation of the Stomach").

As regards position, the stomach may assume a vertical direction, or be located in the thoracic cavity in cases of diaphragmatic hernia. I am not cognizant of *enteroptosis* having been observed in early childhood, although it is not uncommon in young adults, particularly neurasthenic females of spare habit. Any wasting illness by inducing absorption of the fat of the abdominal wall and relaxation of the supports to the viscera predisposes to the displacement of the stomach.

#### CONTRACTION OF THE STOMACH.

The stomach may be abnormally small from a congenital defect, or it may contract as a result of continued vomiting, insufficient feeding, or lack of use and the general atrophy accompanying marasmus. The condition can only be diagnosed by an actual measurement of the gastric contents, while the treatment must be directed towards creating tolerance for a gradually increasing quantity of food, given at regular intervals.

#### DILATATION OF THE STOMACH; HYPERTROPHIC PYLORIC OBSTRUCTION.

Dilatation of the stomach is of more frequent occurrence in children than in adults, although the causes producing this condition most commonly in adults are quite rare in children.

On the other hand, etiological factors of no moment to the adult may produce a marked degree of dilatation in an infant.

**Etiology.**—Dilatation may take place rapidly during the course of an acute gastritis or cholera infantum. It is usually, however, secondary to chronic indigestion or chronic gastritis, as a result of the long-continued distention of the stomach by the slowly digesting food and the gases which generate in these conditions. The obstruction of the pyloric end of the stomach with tenacious mucus is to my mind an important etiologic factor.

The general muscular atony of rickets is a prominently predisposing cause, and in chronic gastritis there is a similar relaxed condition of the muscular wall of the stomach. In the latter condition there is at the same time a certain amount of pyloric obstruction, resulting from the thickened state of the mucous membrane and the tenacious mucous secretion. Pyloric obstruction may result also from inflammatory adhesions; pressure from an abdominal tumor; congenital defect and even malignant disease, although this is quite rare.

*Dilatation not dependent upon pyloric obstruction* is usually the result of giving the food in too large quantities, especially when this becomes necessary in order to satisfy the child's hunger on account of the food being insufficiently nutritious. Feeding the babe with condensed milk is therefore a frequent cause. I have also seen dilatation result in several instances from the use of proprietary foods containing malt in some form, which through fermentation set up sufficient distention to dilate the stomach. In such cases we can withdraw a large quantity of a brownish, offensive fluid from the stomach by inserting the lavage apparatus.

*Hypertrophic pyloric obstruction* is a condition that has received special consideration of late and numerous cases have been reported, although a large number of such have undoubtedly been spasmodic in type. It is therefore necessary to differentiate between true *hypertrophic stenosis* and *pyloric spasm*. Thompson was of the opinion that all of these cases

were primarily spasmodic in origin, the excessive contraction of the pylorus eventually leading to hypertrophy of the muscular coats. This theory, however, does not explain the development of the actual hyperplasia of the epithelial and submucous coats associated with the hypertrophied and infiltrated muscular coats in some of the reported cases, nor does it throw any light on the nature of the spasm. From personal observation I am led to believe that the purely spasmodic cases owe their origin to an excessive acidity of the gastric juice or to the development of irritating foreign products in the gastric contents in sufficient amount to cause over-stimulation and contraction of the pylorus. These are, notably, lactic, acetic and butyric acid. Again, the clogging of the pyloric end of the stomach with tenacious mucus is another factor that must be taken into consideration. The fact that I have been able to control a number of such cases with systematically applied lavage strengthens my views on this point. I have also made mention on a previous occasion (*North Amer. Jour. Hom.*, Dec., 1903) of the fact that in some of my cases of gastric dilatation there was hyperacidity of the gastric contents.

Congenital gastric spasm is therefore a condition of irritability of the circular muscular fibres at the pyloric end of the stomach, existing from the time of birth, in which case it is fair to suppose the pylorus to be congenitally hypertrophied and irritable, or becoming so later as a result of direct irritation. It may be of short duration or persist until hypertrophy sets in, and if the irritation be sufficient to induce inflammatory changes, hyperplasia of these structures ensues. Most of the cases so far reported have been fatal. This is, no doubt, explained by the fact that many of the more benign type of cases escape recognition. I have recently had under observation a well marked case with persistent vomiting; emaciation; dilatation of the stomach and peristaltic waves plainly visible in the gastric area, which ultimately made a complete recovery.

**Symptomatology.**—The stomach is usually symmetrically dilated, with a preponderance of the deformity at the cardiac end. At times the greater curvature may reach below the umbilicus, in which case the normal contour of the organ is much changed, giving it the appearance more of a bagpipe than of a stomach. These extreme cases are, however, rarely encountered.

The physical signs are epigastric bulging and a tympanitic percussion-note, together with splashing when there is fluid in the stomach. Chronic indigestion, belching, vomiting of large quantities of partly-digested food, and often interference with the function of adjacent organs, are the accompanying symptoms.

In *congenital gastric spasm* the stomach is dilated and peristaltic waves can be seen passing across the epigastrium toward the pylorus. In the hypertrophic variety the thickened pylorus can at times be palpated in the epigastric region just to the right of the median line. Vomiting is the cardinal symptom, but it is not always continuous, as the obstruction tends to give way at irregular intervals. At such times milk residue will appear in the stool, while ordinarily the stools are scanty and infrequent, consisting mainly of mucus and bile.

**Diagnosis.** To positively diagnose the condition, an exact outline of the organ and a determination of its capacity must be obtained. (Often we are enabled to percuss satisfactorily the abdomen three to four hours after eating. If this yields unsatisfactory results, half a Seidlitz powder may be administered, or the stomach filled with water. The latter procedure is, however, not entirely without danger in all cases, and is not to be employed haphazard. The lower border should not extend beyond half the distance between the umbilicus and ensiform cartilage; anything below this indicates dilatation.

Extension of the tympanitic note to the left is also important, indicating dilatation of the cardiac end. The phonendoscope is often of service to determine the outline of the

organ by placing its stem in the region of the fundus and observing the changes in sound occurring by stroking the finger in different directions (auscultatory percussion).

Splashing can be obtained where there is gas and fluid present.

*Dilatation of the colon* is to be differentiated by the concave outline of the lower border of the distended area; it is convex in dilatation of the stomach. Besides, the result obtained from the administration of a Seidlitz powder, together with the clinical features of the case, must be taken into consideration. The best results are obtained when the powders are dissolved separately and thus taken, allowing the generation of the gas to take place entirely within the stomach. Naturally for a child a fraction of the powder suffices.

Pyloric obstruction is diagnosed by the persistent vomiting and the absence of true fecal matter in the intestines. Peristaltic gastric waves are pathognomonic of the condition. It is impossible to say whether a case is purely spasmodic or hypertrophic in character unless a sudden cessation of symptoms takes place, or the hypertrophied pylorus can be palpated.

**Treatment.**—The prominent indication for treatment is the indigestion which is present in these cases, being best overcome by a careful regulation of the diet according to the rules laid down in the chapter on "Feeding." Whey and broths are more applicable than milk. Lavage is also of prime importance, especially when there is gastric catarrh or vomiting. The auxiliary measures and remedies mentioned under "Chronic Indigestion" are to be consulted. Naturally, any food capable of undergoing fermentation must be avoided, and even in preparing a milk formula it will be well to decrease the percentage of sugar if gas develops in the stomach.

In pyloric obstruction a surgical operation is the last resort when systematic stomach washing and careful feeding fail to give relief. It is best to restrict the diet to whey, broths, meat juice and albumen water. Milk is not tolerated as the

casein cannot pass through the narrowed pylorus. Malted foods are also contraindicated as they tend to ferment in the stomach.

#### ULCER OF THE STOMACH.

The *round perforating* ulcer of the stomach is very rare, but it has been met with at all periods of infancy and childhood. Its anatomical characteristics are identical with the gastric ulcer of adults, although this variety of ulcer is more frequently found in the duodenum than in the stomach. Cade reports a typical case in an infant two months old, death resulting from perforation. He was able to collect twenty cases from the literature, the ages varying from several hours to thirteen years. Henoeh (*Beitrage zur Kinderheilkunde*, 1861) repeatedly met with the condition clinically. Adriance (*Archives of Pediatrics*) reports a peptic ulcer in the duodenum of an infant ten months old.

A second variety is the *tuberculous ulcer*, which is also rare. A third variety is *follicular ulceration*, which is most frequently found in the newborn. Rotch cites a typical case occurring in a girl one year old (*Pediatrics*).

I have on several occasions encountered follicular ulceration in the stomach and bowels of the newborn dying either of indefinite symptoms or with those of melena (see *Diseases of the Newborn*). The mucous membrane of the stomach is found studded with numerous ulcers of circular outline and about the size of a split pea. They may coalesce and form irregular patches. In some the superficial epithelium alone is destroyed, while others extend into the sub-mucosa, causing considerable hæmorrhage. The stomach contains a tenacious mucus which is stained blackish from the admixture of blood. The greatest number of lesions was found at the cardiac end and at the fundus of the stomach. The colon was at the same time involved in a similar follicular inflammation with ulceration. The small intestine seems to escape, no doubt owing to its alkaline reaction. In this

respect, *aphthous stomatitis* seems to bear a close relationship to the above process, only developing in the mouth when the normal alkaline secretion has become diminished or vitiated—

**Symptomatology.**—Localized tenderness, gastric pain especially aggravated by eating, and the vomiting of blood, or bloody stools, are the cardinal symptoms. These are, however, not always present, and often a positive diagnosis cannot be made. Perforation may occur, resulting in collapse and peritonitis. Colgan cites a case of round perforating ulcer, the presence of which was not suspected until perforation took place.

**Treatment.**—If gastric ulcer be suspected, the child should be put to bed and kept on a milk diet. In case of hæmorrhage food should be withheld, ice may be given, and rectal alimentation instituted. The most important remedies from the clinical standpoint are *Arsenicum*, *Argentum nitricum*, *Mercurius corrosivus* and *Phosphorus*.

#### CANCER OF THE STOMACH.

Malignant disease of the stomach is exceedingly rare during childhood, but it has been met with occasionally. Cullingworth has reported a case of columnar epithelioma occurring in an infant five weeks old. Ashby and Wright (*Diseases of Children*) report the case of a boy aged eight years who died of a columnar epithelioma involving the stomach and duodenum. In this case a tumor could be felt below the edge of the liver, to the right of and on a level with the umbilicus. There was abdominal tenderness and distention, frequent attacks of colicky pains and gradual emaciation.



## CHAPTER VIII.

### DISEASES OF THE LIVER.

The position and relative size of the liver varies with the age of the child ; thus, in the new-born its weight is approximately 4 per cent. of the body-weight, at six months 3 per cent., and in adults 2.5 per cent. Its lower border reaches nearly to the crest of the ilium in infants when in the upright position (*McClellan*), while the upper border reaches the fifth intercostal space in the mammary line, the seventh in the axillary, and the ninth posteriorly. The low position occupied by the inferior border of the liver, in comparison with adults, is not entirely due to the greater development of the organ, but must be explained also by the structural peculiarities of the thorax, for the ribs, by their more horizontal direction, cover the liver to a less extent than the elongated thorax of the adult (*Sahli*, "*Topographische Percussion im Kindesalter*"). During the entire period of childhood the liver edge can be felt extending somewhat below the costal margin.

The *examination* of the liver comprises palpation and percussion. Often the lower edge can be distinctly seen, in emaciated subjects.

In order to *palpate* the liver the child is placed in the prone position and if the abdominal walls be rigid the thighs may be slightly flexed by an assistant. With the tips of the first three fingers of the right hand gently pressed into the abdomen, the examiner feels for the edge of the organ by working from below upward (Fig. 14). Note whether the edge be sharp and regular in contour or whether it be rounded and irregular. Also judge of its consistency, i. e., whether it be softer or firmer than normal.

In the prone position the *deep dulness* reaches up to the



fourth interspace in the mammary line and seventh interspace in the mid-axillary. The *superficial dullness* begins at the upper border of the sixth rib in the mammary line and blends with the cardiac dullness in the parasternal line. In the axillary line it crosses the eighth rib and posteriorly the tenth rib. The percussion stroke should be light in eliciting these boundaries but for the deep dulness it must be moderately strong.

#### JAUNDICE ; ICTERUS.

With the exception of the jaundice peculiar to the newborn, the symptom indicates nothing different from the conditions capable of producing it in adults. *Icterus neonatorum* is a physiological condition and has been described in a preceding chapter.

Jaundice is the result of an obstruction in the gall-ducts or in the common duct. This may happen from a variety of causes.

It may be due to congenital stricture or the accidental entrance of a round worm into the ductus communis choledochus. The pressure of a new growth or the lodgment of gall-stones in the duct are rare occurrences in children. The commonest form of jaundice is *catarrhal*.

*Catarrhal jaundice* depends upon a catarrh of the duodenum and gall-ducts, the swelling of the mucous membrane, together with the production of tenacious mucus, inducing the obstruction. It is most frequently seen after the third year of life. The accompanying symptoms are headache and lassitude, anorexia, diarrhoea, or, more commonly, constipation, the stools being light in color and very fetid ; high-colored urine, due to the presence of bile-pigments, and occasionally slight fever at the commencement of the attack. The liver is slightly enlarged (Fig 14). A marked reduction in the pulse-rate, which is the case in adults, does not take place at this age.

This affection is not uncommon in children ; some show

the disposition to jaundice from infancy. Indigestion and constipation, often obstinate, are as a rule associated and sometimes round worms are abundantly present, marked improvement following upon the exhibition of santonin. A distinct gouty family history may exist.

#### CHOLELITHIASIS.

Although cholelithiasis is rare during childhood, still we are likely to encounter it at times, and must not lose sight of this fact in the differential diagnosis of painful abdominal affections.

I recall the case of a child four years old which presented the history of an attack of gall-stone colic three months prior to the time I saw it; and in a three-year-old boy suffering with violent abdominal pains I was able to demonstrate minute biliary calculi passed with clay-colored stools several days after the attack. Since then I have noted two other such cases. Many cases are undoubtedly overlooked, being looked upon as gastralgia or intestinal colic. A condition from which cholelithiasis must be differentiated is appendicular colic, which, according to Van Lennep, is common in children, owing to the patency of the opening to the appendix.

#### ACUTE YELLOW ATROPHY.

This is one of the rarest diseases of childhood, only about fifteen cases being on record, according to Lanz (*Wiener Klinische Wochenschr.*, 1896). Fison (*Lancet*, July, 1897) reports a case in a girl twelve years old, giving detailed autopsy findings. It must be considered in the differential diagnosis of obscure serious ailment by which the nervous system is profoundly affected. Simulating a simple catarrhal jaundice in the beginning, the symptoms gradually assume a most alarming type, delirium, uncontrollable vomiting, dilatation of the pupils, coma and convulsions developing. The urine contains bile pigments, leucin and tyrosin; in Lanz's case albumin and acetone were also present. The tempera-

ture rises with the progress of the disease and the spleen enlarges. Other symptoms which may be observed are progressively-increasing jaundice, slight œdema of the extremities, atrophy of the liver, ecchymoses and bleeding from the gums. A fatal termination usually takes place within a few weeks from the onset, or, exceptionally, within a few days. The liver appears wrinkled and of a yellow-ochre tint, the lobules becoming indistinct. The hepatic cells are filled with granules and the tissue is largely replaced by granular and fatty debris contained in a reticulated homogenous structure.

#### CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver is much rarer during childhood than during adult life, as alcoholic excess, the chief etiological factor producing this affection, is only in exceptional cases operative at this age, the other cases being regarded as syphilitic and tuberculous, although the eruptive fevers are considered by Laure and Honorat (HOLT) capable of producing interstitial changes in the hepatic glandular structure. Morse (*Boston Med. and Surg. Jour.*, Sept. 11, 1902) states that in hospitals the disease has been encountered one in twenty thousand cases. He looks upon intestinal auto-intoxication as a probable cause. In early infancy it is seen in association with obliteration of the bile ducts (congenital) as biliary cirrhosis. Syphilitic cases and those peculiar to India are also seen in early infancy.

The toxins of tuberculosis may cause cirrhosis with tubercles being present. Organic heart disease may also be followed by interstitial changes in the liver.

The hypertrophic variety is most frequently met with, especially when traceable to syphilis. Only when the interstitial changes are pronounced will symptoms referable to the liver be induced, in which case the course is the same as in adults. More commonly, however, the cirrhosis is not suspected, being either masked by the symptoms of the exciting cause (congenital syphilis, tuberculous peritonitis, etc.), or

there are insufficient symptoms to give a distinct type to the disease.

**Treatment of Hepatic Diseases.**—In selecting the diet for hepatic disturbances, we must consider the digestive as well as the assimilative functions of the liver. The rôle of the biliary secretion in the digestion of fats renders it necessary to cut down the percentage of fat in the food, as intestinal indigestion or a fat diarrhoea will result from an excess of this food when the bile is deficient in amount. Carbohydrates, being stored up in the liver, must also be given sparingly. A milk formula, with a reduction in the percentages of fat and lactose, will be indicated in infants; older children may be put on the ordinary milk diet, together with fresh, succulent vegetables, light meats, and stewed fruit. Water must be administered abundantly.

*Acute yellow atrophy* is universally recognized as being invariably fatal, and symptomatic treatment is all that can be instituted to ameliorate conditions as they arise. *Cirrhosis* has been benefitted by anti-syphilitic treatment, and the alcoholic form can be arrested if taken in time.

Remedies which will be indicated by the presence of *jaundice*, and evidence of *gastro-duodenitis* or *acute intestinal catarrh*, are *Berberis*, *Bry.*, *Calc. carb.*, *Cham.*, *Chelid.*, *China*, *Digit.*, *Gels.*, *Leptandra*, *Merc.*, *Myrica cerif.*, *Nux vomica*, *Podoph.*, *Pulsatilla*, *Sulph.* Of these, *Bryonia*, *China*, *Mercurius vivus* and *Nux vom.* are the most useful and most frequently indicated, mainly from their characteristic symptoms referable to the tongue, thirst, appetite, stool, etc.

The tendency to the formation of *gall-stones* is markedly influenced by *China*. For the painful symptoms, *Bell.*, *Bry.*, *Cham.*, *Nux vom.* and *Calc. carb.* are occasionally of use, although hot fomentations, and in extreme cases an anodyne or anæsthetic, will become necessary. A mild saline aperient, the best of which is the Carlsbad Sprudel Salt, is of great value to prevent the accumulation of mucus in the bile ducts and avoid the baneful effects of constipation.

## CHAPTER IX.

### DISEASES OF THE INTESTINES.

During infancy the process of digestion takes place most prominently in the small intestines, as the stomach is not fully developed at this period of life, and must be considered more as a reservoir for food than the principal organ of digestion, although pepsin, rennin and free hydrochloric acid have been found in small quantities in the stomach of the new-born. The important change taking place in the milk while in the stomach is its coagulation by rennin. The action of rennin, which is an enzyme, is to coagulate, or clot the *caseinogen* (the original proteid) into *casein*. The next step is the action of the hydrochloric acid of the gastric juice upon the coagulated milk, the product being *paracasein chlorid*. A portion of this is subsequently attached by the pepsin. The curdling of milk through souring is purely a precipitation of the proteid and the addition of an alkali will cause it to be re-dissolved. This does not take place when rennin has coagulated the casein. The bulk of the work of digestion is therefore thrown upon the intestinal tract, gastric digestion being, so to speak, a preparatory step. For this reason disturbances are more frequently found here than in the stomach.

The intestinal tract is relatively larger in children than in adults, being six times the body-length in the new-born, while in the adult it is but four times (BENEKE). The sigmoid flexure is notably long, constituting one-half the length of the greater bowel.

Intestinal digestion is very active in the normal infant owing to the large amount of bile secreted, which saponifies the fats and stimulates peristalsis. Trypsin and steapsin are also actively secreted, the former peptonizing proteids and the latter emulsifying fats. After the third month the diastatic ferment

of the pancreatic juice (amyllopsin), which has the power of converting starch into sugar, makes its appearance, so that it becomes possible for the infant to digest farinaceous foods. This form of digestion is, however, not fully established until the time of the eruption of the teeth, before which time it is unwise to use starchy food unless predigested. The muscular coats of the intestines are poorly developed at this period of life.

A rational understanding of the diseases of the intestinal tract presupposes an intimate knowledge of the character and composition of the stools in health and in disease. Without this knowledge it is impossible to diagnosticate the various disturbances of the functions of the intestine or pathological lesions here found. Besides, we will often fail to understand the true nature of an apparently obscure constitutional disorder if we neglect to investigate the intestinal discharges for evidence of intestinal parasites, which frequently affect the general health to a marked degree.

Unfortunately, the examination of the fæces impresses the average physician as a repulsive procedure, and the benefits to be derived from such an examination are held inadequate compensation for the unpleasantness of the task involved. But with proper technique this is not the case, and especially so in infants. In pædiatric work there is positively no excuse for neglecting such an examination whenever it is called for.

In older children a specimen of fecal matter is best obtained by inserting a piece of glass tubing with rounded ends into the rectum, for a distance of about three inches, and allowing it to remain in place five minutes. By the end of that time the peristaltic action of the rectum will usually have filled the tube. In infants a freshly soiled diaper can, as a rule, be obtained without difficulty, although, when we wish to be absolutely certain that no urine is admixed with the stool, we will have to resort to the tube.

*The Normal Infantile Stool.*—Shortly after birth the infant

passes three to four stools, consisting of meconium,—a thick, tarry substance, representing the biliary and mucous secretions that have collected in the intestinal tract during intra-uterine life, besides epithelium and particles of vernix caseosa and hairs. Following this the normal milk stools make their appearance.

The normal milk stool is of a golden-yellow color and of a thick, smooth, pasty consistency, without definite formation. The odor is slightly sour, not offensive, and the reaction is acid. A large percentage of water is present, so that a ring of moisture surrounding the fecal matter forms on the diaper. This ring normally extends for a finger's breadth around the stool; any considerable increase in moisture beyond this point is abnormal. In the early months of infancy there are from three to four stools in twenty-four hours; by the end of the first year the number is decreased to one or two.

The first abnormal condition to be observed in intestinal disorders is an *increase in the size and in the frequency of the bowel movements*. This means intestinal indigestion, or dyspeptic diarrhœa. When we pause to reflect that the main work of digestion, and practically all of assimilation, takes place in the small intestines of the infant, we must be impressed by the fact that such a condition may be the forerunner of most serious consequences.

In the dyspeptic stool we discover, first of all, particles of undigested milk, "Milchdetritus," almost universally and erroneously designated "curds." Far from representing mere particles of undigested casein, their composition is most complex and variable. While casein in greater or less proportion may be present in these clumps of fecal matter, still their composition is chiefly of fat, together with fatty acids and lime-salts. Indeed, in some forms of diarrhœa the fat percentage is so high (30 per cent. to 50 per cent.) that the condition has been designated "fat diarrhœa" (Biedert; Demme).

Together with the above alterations in the character of the stool, there is also a change in the *color*, manifesting itself as



an admixture of green. The green color is due to the presence of biliverdin. Several explanations for its presence may be offered. In the first place, bacterial changes in the intestinal tract, by which the bilirubin is oxidized into biliverdin, may change the color of the stool to green already in the intestinal tract. In other cases there is simply an excess of bile, which is promptly oxidized on exposure to the air, the stool thus becoming more and more green as it stands.

Again, as Pfeiffer (*Jahrbuch für Kinderheilk.*, 1888) points out, the green color in the stool depends upon the action of an alkali on the bilirubin and does not signify acid fermentation, as was formerly taught. The important point to remember is that while an alkali changes the color to green, an acid does not convert it back again to yellow. Therefore, an alkaline zone must exist somewhere in the intestinal tract—the alkali being most likely derived from the pancreatic juice. The reason for its excessive action is either feeding milk in too large quantities, thus neutralizing the gastric contents completely, or hypo-acidity of the gastric juice. After the intestinal contents have passed this alkaline zone, they may again become acid through the action of the *bacillus lactis ærogenes*.

Another cause of green stools is the chromogenic *bacillus* described by Le Sage. This is rarely present.

The admixture of green and yellow, together with the white particles of "Milchdetritus," produces the characteristic appearance described as chopped eggs and spinach.

A further abnormal change in the stool is an *increase in its fluid elements*. Blood serum is always freely poured out in inflammatory conditions of the intestinal mucosa, and in cholera infantum the evacuations consist essentially of serum.

*Increase in the Number of Stools.*—An increase in the number of stools indicates either that the food is being hurried through the intestinal tract in an undigested state or that an inflammatory condition has supervened. Increased peristalsis is an important factor in both conditions. In dyspepsia there



may be from four to six stools daily. In inflammatory conditions of the upper bowel the stools are large, increased from six to eight daily and, as a rule, expelled with considerable flatus. Gastric symptoms are a frequent accompaniment. On account of the fermentation taking place in the bowels the abdomen is distended. When the lower bowel is affected the stools are smaller in size and more frequent, while involvement of the rectum produces tenesmus that may practically be continuous. In such cases only a small amount of faecal matter is passed, but considerable mucus and usually some blood are present.

*Decrease in the Number of Stools.*—An abnormal decrease in the number of stools is designated constipation, when due to deficient peristalsis, insufficient or improper food, or abnormal dryness of the mucosa. The various forms of bowel obstruction cannot be considered here.

*Mucus.*—Mucus is found in insignificant amount in both normal and dyspeptic stools, but in inflammatory states it is always present in considerable quantity. In fact, in catarrh of the intestine, it may be the chief, if not the sole, constituent of the movements.

The character of the mucus offers most valuable data in the recognition of the seat of the lesion in inflammation of the bowel. When thoroughly admixed with the other elements of the stool and stained with bile, it comes from the small intestine. Under these circumstances gas usually accumulates in the intestines and the abdomen becomes distended.

Mucus coming from the large intestine is more abundant, not so intimately admixed with the fecal matter, and not thoroughly bile-stained. The mucus secreted from an inflamed rectum is passed in clear, jelly-like lumps, blood-streaked.

*Blood.*—Profuse hæmorrhage from the intestine most commonly originates in either tuberculous or typhoid ulcers. Hæmorrhoids are rare in children, but rectal polypi are not uncommon.

Blood from the small intestine gives the stool a dark, tarry appearance. In the newborn, intestinal hæmorrhages are at times encountered, the blood coming from folliculous ulcers in the stomach or large intestine (*melenæ neonatorum*). Blood passed in fresh clots comes from the rectum or lower part of the colon. Small quantities thoroughly admixed with the stool in diarrhœa originate in capillary hæmorrhages.

*Color.*—The color of the stool is affected in a pronounced manner by certain drugs and by the food. As above stated, the normal milk stool is of a golden yellow. Excessive amounts of fat may give it a grayish color, while excess of proteids usually brings about greenish discoloration. Barley-water and meat-juice tend to give it a brownish color. In obstructive jaundice the stool becomes clay-colored. *Calomel* produces a decidedly green stool, loose in character. *Bismuth* and *Iron* cause the stool to turn black.

*Chemical Examination.*—The chemical examination of the fæces has yielded data of the highest clinical importance. In this connection the *odor* may be considered, as it depends upon chemic changes in the food induced mainly through the agency of bacteria.

The sour odor of the infantile stool depends upon the presence of fatty acids and to the action of the *bacillus lactis ærogenes* upon the lactose, which is transformed into lactic and butyric acids. Under pathological conditions, acetic, formic and other organic acids may appear.

A foul odor indicates decomposition of proteids into tyrosin, indol, skatol and phenol. This is encountered in the severer forms of infectious diarrhœa.

The *reaction* is acid in the majority of diarrhœas. Baginsky states that it is likely to be alkaline when the odor is foul, indicating the presence of ammonia compounds from decomposition of proteids. From extended personal observations I have come to the following conclusions:

In dyspeptic diarrhœa, or in affections of the upper intestinal tract, the reaction is acid. This, no doubt, depends

upon the fact that in the small intestine the *bacillus lactis aerogenes* predominates. Besides, in these affections, mucus and serum are not as abundant as in affections of the lower tract.

Stools from the lower tract are, as a rule, alkaline. Here the *bacillus coli* predominates and proteid decomposition is most active. Moreover—more mucus and serum enter into the composition of the stools from this region. I have invariably found that where mucus was abundant the reaction was either alkaline or neutral. Blood serum being alkaline naturally tends to render the stool so.

Neutral stools are frequently seen. A combination of causes seems to be active here.

*Bile pigments* are increased in catarrhal conditions, biliverdin predominating. Stercobilin (identical with urobilin), the coloring-matter of the stools, is not found in any considerable amount owing to the absence of putrefactive changes, but hydrobilirubin—a reduction compound of bilirubin—is found when fermentation with the liberation of hydrogen occurs. It can be readily detected by the corrosive sublimate test.

Blauberg (*Experimentelle u. kritische Studien Ueber Säuglingsfæces*, Berlin, 1897) has made the following observations in his careful work in this line :

The green color of the stools is due to biliverdin, which not only develops after exposing the fæces to the air, but which is always present in the slightest digestive derangement. He is inclined to think that certain ferments play an important rôle in its production. The sour odor depends upon free fatty acids and butyric acid.

The amount of nitrogenous compounds averages about 4 per cent.

Fat is found in considerable quantity in the fæces during the early weeks of infancy, but under normal conditions a decided decrease in this ingredient occurs after the seventh and eighth days. An actual fat diarrhœa may occur in the new-

born, indicating that it must accommodate itself to breast milk as well as to any other food. Chapin (*Archives of Pædiatrics*, July, 1903) expresses similar views, basing his argument upon a study of the evolution of mammals. He writes as follows: "While the stomach of an infant is formed at birth, its function is not developed. Strictly speaking, then, an infant has no stomach at birth, as it does not secrete pepsin and hydrochloric acid, but a dilated sac that develops into a true stomach during the suckling period."

Lactic acid, fatty acids and iron are present in larger amounts in the fæces of breast-fed than in bottle-fed infants.

When cow's milk is fed there is a larger proportion of fat, nuclein, lime-salts and phosphoric acid.

The gases represent the products of lactose fermentation, together with some swallowed air and CO<sub>2</sub>. Normally, they are never foetid.

Diastatic and invert ferments are normally present.

*Microscopical Examination.*—If a bit of normal fæces be placed upon a slide with a drop of normal saline solution and examined with a low power, we will not find much of interest. Small particles of nitrogenous matter, fat globules and crystal of fatty acids, traces of mucus, a few epithelial cells and *débris* constitute the chief elements. Animal parasites are absent. The normal bacteria will be considered further on.

When the child is artificially fed, the findings in the microscopical field will depend upon the nature of the food administered. Under these conditions it is also not infrequent for animal parasites to show themselves.

The various cereals used in infant feeding leave a considerable amount of indigestible vegetable *débris* in the stools, representing the cellulose walls of the cells in which the starch-granules are contained. From an examination of a large number of diarrhœal stools in which barley-water and other cereals were used as a diet, I have been led to believe that these foods are not without their drawbacks in

inflammatory states of the intestinal mucosa. In this belief I am still further strengthened by the following findings, which indicate the microscopic appearance of the different cereals under different conditions:

*Barley-Water Made from the Grain.*—(In these examinations a two-third-inch objective and a one-inch eye-piece were used.) The field contains broken-down starch-granules and homogeneous starchy material, together with a large amount of cellulose detritus, wooden in appearance. The bits of cellulose structure represent clusters of from ten to twenty starch-granules, and some are visible to the naked eye.

*Barley-Water from Patent Barley-Flour.*—No starch-granules, but homogeneous starch material, together with abundant cellulose detritus, slightly finer than above.

*Barley-Flour Mixed With Cold Water.*—Starch-granules and cellulose detritus, some visible to the naked eye.

*Wheat-Flour, Boiled.*—Broken starch-granules and homogeneous starch material. Clusters of swollen starch-granules in cellulose sheaths and cellulose detritus.

*Wheat-Flour Mixed With Cold Water.*—Starch-granules free and in clusters, with envelope of cellulose.

*Rice-Water Made from the Grain.*—Starch-granules broken down and in solution. There is some cellulose, but it is not so coarse nor as abundant as in barley or wheat.

*Arrowroot Mixed with Cold Water.*—Starch-granules free from foreign admixture.

From the above it will be seen that the blandest solution on which the infant can be fed is arrowroot water, after which comes rice-water. Wheat and barley both contain too much cellulose, particularly barley. In health this is no disadvantage, but under abnormal conditions it must be taken into consideration.

*Charcot-Leyden Crystals.*—The flat, needle-like crystals first discovered in the sputum of patients suffering with bronchial asthma are also found in the fæces quite constantly in cases of anchylostomiasis. Not so constantly, but quite fre-

quently, they are encountered in association with tape-worm, ascarides, oxyurides and in amœbic dysentery. (Amberg; Simon.) On account of their close association with eosinophilic leucocytes they have been termed leucocytic crystals. These leucocytes and their free granulations can be demonstrated in such fecal matter by staining with eosin.

*Blood and Pus.*—Blood and pus-corpuscles are at times found in the fæces when the naked eye does not suspect their presence. In such cases it is well to stain for tubercle bacilli, as tuberculous ulceration may be the source of these elements. It has been stated that the bacillus acidophilus of Moro possesses staining properties similar to Koch's bacillus, but I have not been able to satisfy myself that mistakes in diagnosis could thereby arise.

*Parasites.*—In the fæces of children under mixed feeding, Pagliari (*Jahresbericht über Thierchemie*, 1894) found the eggs of parasites in 90 per cent. of cases. They represented ascarides, trichocephalus and tænia solium. The eggs of the oxyuris are not found in the stool. The trichomonas is a protozoön of spindle-shape, with four flagellæ at its anterior pole, and is of no pathological significance. It is thought to be identical with the trichomonas found in the vagina and in the urine. In examining for parasites and ova it is well to add a drop of Grassi's fluid (aqueous solution of iodine with potassium iodide) to the fecal matter.

*Amœb. Coli.*—This organism was discovered in the stool of dysentery patients by Lösch in 1875, but its true relation to the disease was first established in 1885 by Kertulis. In America, Osler was the first to demonstrate the amœba in an hepatic abscess complicating amœbic dysentery.

Amberg (*Johns Hopkins Hospital Bulletin*, December, 1901) reported five cases of amœbic dysentery in children ranging from three to five years. The amœba are motile and contain red blood-corpuscles. They may be stained with a watery solution of toluidin blue, which does not kill them for from three to four hours. If the amœbic movements are not discernible, the slide should be warmed.



*Helminthes*.—*Oxyurides* can often be obtained by means of the rectal tube, but their eggs are not deposited in the feces. The eggs are smaller than those of the ascaris and are oval in shape. The ascaris deposits the eggs directly into the intestine. They are yellowish-brown in color, almost round, from 0.05 to 0.07 mm. in diameter, and surrounded by an irregular albuminous shell. (See illustrations under "Intestinal Parasites").

The ova of the *Uncinaria Americana* (hook-worm) are illipsoids, 64 to 76 micromillimeters long by 36 to 40 broad, in some cases partially segmented, in others containing a fully developed embryo. Their color is grayish, like that of a steel engraving. (Stiles, *Bull. No. 10, Hyg. Lab. U. S. Pub. Health and Mar. Hosp. Serv.*, Washington, February, 1903.)

*Tænia saginata* has elliptical ova of a brownish color with a distinct vitelline membrane. A double contour and striæ may be demonstrated under high magnification. *Tænia solium* is rare in this country. The ova are surrounded by a thick, striated membrane, and the hooklets of the embryo can be seen within the ovum.

*The Bacteria of the Intestinal Tract*.—The normal bacteria of the intestinal tract are represented chiefly by the bacillus lactis ærogenes and the colon bacillus. The former is found mainly in the upper intestinal tract, while the latter predominates in the large intestine. The duodenum is comparatively free from bacteria under perfectly normal conditions. The bacillus lactis ærogenes disappears from the stools as soon as the milk diet is dropped. Moro has described a bacillus which he calls the bacillus acidophilus, and which, according to his investigations, normally exceeds all other micro-organisms in the stools of breast-fed infants. He has isolated it from the nipple of the human breast and from the milk. Under abnormal conditions it becomes diminished and the colon group predominates. The chief characteristic distinguishing it from the colon bacillus (including the typhoid bacillus and Shiga's bacillus) is the fact that it does not decolorize by Gram's method.

By Escherich's stain it therefore stains blue, while the colon group is stained red. Escherich (*Die Darmbakterien im Säuglingsalter*, 1886) was of the opinion that under normal circumstances most of the colon bacilli resisted the *Iodine* solution and did not lose their stain, while in diarrhœal affections they were decolorized. This view, however, has been controverted by Moro's investigations. (*Wiener Klinische Woch.*, No. 5, 1900.)

Nevertheless, Escherich's stain is of the greatest practical importance, as it demonstrates the exact proportion between normal and abnormal bacteria in the infantile stool. It is carried out as follows :

A cover-glass preparation of the stool is fixed in the flame of a Bunsen burner and stained for a few seconds with aqueous gentian violet plus aniline oil and blotted ; it is then immersed for a few seconds in aqueous *Iodine* solution and blotted ; decolorized with a mixture of equal parts aniline oil and xylol, washed in xylol and dried. The specimen is now counter-stained with alcoholic fuchsin, washed with water, dried and mounted in Canada balsam. The formulæ for the stains are :

1. Aqueous solution of gentian violet, 5:200. Boil for half an hour and filter.
2. A mixture of absolute alcohol and aniline oil in the proportion of 11:3.
3. Mix No. 1 and No. 2 in the proportion of 85:15. This represents the stain, which will only keep for two or three weeks.
4. A solution of *Iodine*, one part ; *Potassium iodide*, two parts ; water, sixty parts.
5. Concentrated alcoholic solution of fuchsin, diluted with an equal volume of absolute alcohol.

With this method, normal and abnormal stools can even be distinguished macroscopically, by the preponderance of the blue color in the former and the red in the latter. When streptococci are present, as is the case in grave inflammatory



lesions of the intestinal mucosa with resulting infiltration and necrosis of the tissues, they retain the blue color, but are readily distinguished from the bacilli by their form

While the colon bacillus and the bacillus lactis aerogenes are normally saprophytes, still it has been clearly proven that both, especially the colon bacillus, may, under certain conditions, assume pathogenic properties.

The proteus vulgaris is often found in the stools of artificially-fed infants, and when active produces a foul odor. It is usually regarded as non-pathogenic. The chief interest attached to it is that at one time pure cultures were used for therapeutic purposes as it was found that the colon bacilli could not exist side by side with the proteus.

It is characterized by its variable forms and is decolorized by Gram's method.

*Shiga's Bacillus*.—Since the investigations of Duval and Bassett at the Thomas Wilson Sanatorium in Baltimore, during the summer of 1902, which resulted in the surprising discovery that the bacillus dysenteriae of Shiga was the etiological factor in the series of cases of summer diarrhoea under observation, this organism has come to occupy the most prominent rôle in the bacteriology of the intestinal tract of children. Duval had previously been engaged in studying the acute dysenteries of adults under Flexner, of the University of Pennsylvania, and his work was therefore immediately accepted as authentic. In an address before the medical association of New York City (October, 1903), Flexner commented upon the work of Duval and Bassett, stating that while these investigators were not prejudiced in the belief that the bacillus of Shiga was a distinctive germ of summer diarrhoea, still all other organisms present resisted the test applied to them. Cultures were made and the agglutination test employed. In over forty cases was the bacillus isolated. Since then the Shiga bacillus has been isolated repeatedly from the stools of children suffering with acute diarrhoea, both here and abroad.

The bacillus is a short rod with rounded ends, and is

slightly motile. Vedder and Duval claim to have demonstrated flagellæ. It does not produce spores, and, like the other members of the colon group, decolorizes by Gram's method. Like the typhoid bacillus, it possesses distinct agglutinating properties with the diluted blood serum from an infected individual. On the strength of this fact it was hoped that a curative antitoxic serum might be evolved. Its growth is slower than that of the colon bacillus, and in a soft jelly it forms a perfectly spherical colony, while the typhoid bacillus forms threading colonies, and the colon bacillus a collection of small colonies. It is more difficult, however, to distinguish it from the paratyphoid bacillus. (Dunham, *N. Y. Med. Record*, Feb. 28, 1903.)

It is best isolated as follows: Grow on agar plates at 37° C., and mark with a pencil the colonies appearing at the end of twelve hours. These are usually colon bacilli. The ones appearing later should be transplanted to glucose-agar fermentation-tubes in order to differentiate the gas formers. The agglutination reaction is possible with a 1-to-50 dilution of the blood serum of the patient afflicted. In fresh bouillon cultures the bacillus is motile during the first eight to twelve hours. It has but slight resistance to heat and antiseptics. (Muir and Richie, *Manual of Bacteriology*, 1903.)

#### SIMPLE DIARRHŒA; ACUTE INTESTINAL INDIGESTION.

**Etiology.**—Owing to the functional and structural peculiarity of the stomach in infancy, the main work of digestion is thrown upon the intestinal tract, and for this reason intestinal disturbances are relatively more frequent than gastric during this period. Even in the case of gastric indigestion diarrhœa is usually a secondary manifestation, owing to the entrance of the unsuitable or excessive quantity of food into the intestines when not promptly vomited. Although the dyspeptic variety of diarrhœa is the most frequent form encountered, and, as has been said before, is due to overfeeding or to the use of improper food, such as breast-milk too rich in

proteids or fat, or vitiated by maternal ill-health, and in the case of hand-fed infants, the use of insufficiently-diluted cow's milk, swill milk, starchy food in early infancy, etc., still there are other influences capable of inducing a looseness of the bowels, to which belong mechanical irritation from the ingestion of a foreign body or irritating substances and nervous influences, such as chilling of the surface of the body, hot weather, fright, dentition and idiopathic irritability of the intestinal tract.

Predisposing factors are important; among them are previous attacks of intestinal catarrh, anæmia, chorea, malnutrition, rickets, tuberculosis and syphilis. Physiological predisposition is most active during infancy; pathological during childhood.

**Symptomatology.** — The cardinal symptoms are colicky pains, flatulence and dyspeptic stools. Moderate fever is usually present. The early symptoms are pain and flatulence, with an increase in the number of stools. The number is seldom greater than five to six daily, and the watery element and amount of mucus are not increased, but there is present undigested food in considerable quantity, and in the case of a milk diet, white flakes consisting chiefly of fat. Curds of casein are often present, but not to the extent of the fat, and are distinguished from the latter by their tougher consistency and insolubility in alcohol and ether. At times the percentage of fat in the stools reaches as high as 50 per cent. and over, in which case it is known as *fat-diarrhœa* (DEMME; BIEDERT). In case of excessive proteids the color is generally green. The green color is due to the presence of biliverdin.

The "spinach and eggs" appearance is due to the intermixing of the fat flakes with the yellow and green elements of the stool. Beside the biliary, a *bacillary green diarrhœa* is recognized, in which the color is produced by a chromogenic bacillus, and which is supposed to be more prevalent in the later period of infancy than the former (LE SAGE)

The duration is short and fever is slight or altogether ab-

sent. Should the condition, however, become prolonged through neglect of proper hygienic methods and lack of medical attention, the foundation for the development of rickets is laid. Fat-diarrhœa in its aggravated form often proves fatal, owing to its dependence upon serious pancreatic or hepatic disease. Demme reported nine such cases.

**Diagnosis.**—The short duration, the inconsiderable fever or absence of fever, and the character of the stools differentiate simple diarrhœa from *cholera infantum* and *entero-colitis*. Neither are the watery elements markedly increased, as in the former, nor do we find present abnormal quantities of mucus and other constituents, such as blood and round cells, frequently found in the latter. The characteristic color and the presence of undigested food particles are the pathognomonic symptoms. In hot weather infants commonly have *watery, yellowish stools* due to the enervating effect of heat and humidity upon the nerves controlling the secretions and movements of the intestines.

The *bacillary form* is differentiated from the biliary by adding *Nitric acid*, which decolorizes the former and changes the latter to violet. The transition of a simple diarrhœa into an inflammatory or choleraic affection may take place.

The diarrhœa ushering in some of the *acute infectious fevers* can only be identified by the ultimate appearance of the symptoms peculiar to the affection in progress.

**Treatment.**—Based on our knowledge of the predisposing and exciting causes of simple diarrhœa, the treatment must be mainly dietetic. Starchy foods, excepting in weak solution, such as barley water, or in dextrinized form, should not be administered until the function of the salivary glands and pancreas has become well established, which is not before the sixth month, and is indicated by the eruption of the teeth.

Infants whose digestive powers are naturally weak should be put on a milk-diet whose formula shall closely approximate human milk in composition; in some instances it may even be necessary to reduce one of the component parts of

the formula below this percentage, as indicated by the symptoms. When the proteids are not properly digested there will be vomiting of curds and the presence of the same in the stool; the fat may also be found in the stool in excess, or produce vomiting. Flatulence points to fermentation of the sugar. The usual mistake is to begin with a milk too highly concentrated, thus entirely upsetting the infant's digestion. It is always better to begin with a weak milk and gradually increase the strength.

In breast-fed infants it may become necessary to analyze the mother's milk, and regulate her diet and exercise according to the indications thus furnished. Regularity in feeding must be insisted upon.

During an attack it is advisable to withhold the usual food for twelve to twenty-four hours, as necessary, and administer simply boiled water, albumen-water or a thin barley-water; then sterilized milk and lime-water (barley-water three parts, milk one part, lime-water one part) may be tried.

In the diarrhœas accompanying teething, or those of a neurotic type, such remedies as *Aconite*, *Belladonna*, *Chamomilla*, *Gelsemium*, the *Calcarea*s and *Pulsatilla* are the ones usually indicated. In fat-diarrhœa *Pulsatilla*, *Hepar* and *Magnesia carb.* have proven most useful in my hands.

In the class of cases in which the intestinal tract seems upset by the food, *Nuxvomica* and *Lycopodium* are most valuable remedies. They are the remedies recommended by Hughes (*Manual of Therapeutics*), and I can vouch for their usefulness. In the non-inflammatory cases, *Nux* in low dilution, where enteritis has been set up, *Lycopodium* 30th (HUGHES).

*Mercury* I prefer to *Lycopodium* in diarrhœa when there is an abundance of mucus. *Mercurius vivus* 3x trit. will check most cases of diarrhœa promptly. When the stools are grass green *Merc. dulcis* is better indicated. *China* is useful in the diarrhœa of hot weather.

*Aon.*—In the beginning; after exposure to cold or during hot weather; thirst, fever and restlessness.

*Ars.*—Diarrhœa after ice water, ice cream, etc.; usually with neuralgic pains in abdomen.

*Aloes.*—Flatulence and rumbling in lower bowels; large quantities of gas escape with stool.

*Bell.*—During hot weather and dentition; face flushed, abdomen distended, colicky pains; cerebral symptoms; skin more moist, and child less restless than in *Acon.*

*Bry.*—Sudden changes in the weather, especially when there are hot days and cold nights. Diarrhœa worse mornings, painful, aggravation from motion.

*Calc. carb.*—Dentition; vomiting and diarrhœa. Stools grayish, chalky, offensive, undigested; worse in afternoon and evening.

*Calc. phos.*—Dentition delayed; recurring attacks; stools green, with flatus; abdomen flaccid.

*Cham.*—Dentition; painful, excoriating diarrhœa, looking like spinach and eggs. Child cross and irritable.

*China.*—Undigested stools; flatulent colic, or painless stool with much fermentation. Anæmia and prostration; loose yellowish stools in hot weather.

*Colocynth.*—Pain relieved by firm pressure.

*Cupr. ars.*—Diarrhœa, with abdominal pains and vomiting. Also when there are no special symptoms for any other remedy.—(GOODNO.)

*Dulcamara.*—During cold, damp weather.

*Gels.*—Diarrhœa from fright, in older children of nervous temperament.

*Ipecac.*—From unripe fruit, sweets, sour or fat articles of food; vomiting, with coldness of extremities and pale face, even convulsions; stools green as grass.

*Hepar.*—Stools white and foetid, or clay-colored (*fat diarrhœa*); generally sour odor both of stool and child. Worse after eating and drinking cold water; sour eructation; hepatic derangements.

*Mag. carb.*—Stools green and frothy, like frog-pond scum, containing tallow-like lumps. Sour odor; colic relieved by stool; *fat-diarrhœa* and *lienteria* of sucklings.



*Mercurius*.—"It is the better indicated the more widely is the departure from the natural color of the motions, and the more slimy they are."—(HUGHES.) Diarrhœa with teething; measles; marasmus, etc.

*Nux vomica*.—Acute intestinal indigestion. In the beginning before inflammatory reaction has been set up and the stools are composed of undigested food only. It corresponds to the stage in which *Castor oil* or *Calomel* is usually employed to clean out the gut.

*Podophyllum*.—Diarrhœa of changeable character, especially as regards color. Usually thin and painless, with much gas. Prolapsus ani.

#### ACUTE INFECTIOUS DIARRHŒA.

Infectious diarrhœa differs from simple diarrhœa in the fact that micro-organisms play the most important role in its production. A simple diarrhœa indicates merely a disturbance in the peristalsis and intestinal secretion or in the chemistry of digestion. In infectious diarrhœa, however, we have to deal with either the direct action of germs or of their toxins upon the intestinal mucosa.

Certain bacteria are capable of producing toxins in milk. When milk so contaminated is fed to an infant it will produce an attack of *cholera infantum*, or *acute milk-infection* (Vaughan). The ordinary putrefactive bacteria, notably, the *proteus vulgaris* and the *colon bacillus*, are the ones most active in this direction. Putrefactive changes may also take place in the gut, setting up similar symptoms. Under these circumstances we speak of *acute fermental diarrhœa*, or *acute gastro-enteric intoxication* (Holt). The *bacillus lactis aerogenes* when present in abnormally large numbers is very apt to set up a fermental diarrhœa characterized by frequent, acid, foul-smelling stools.

Lastly there are a number of bacteria that exert a specific action upon the intestinal tract, setting up *ileo-colitis* (inflammatory diarrhœa) of varying intensity. It was formerly

taught, notably, by Escherich, that under abnormal conditions, especially favored by the action of continued hot weather and high humidity, the normal bacteria of the lower bowel (*colon bacilli*) acquired pathogenic properties and so set up an ileo-colitis. *Streptococci* were also found quite constantly associated with severe ileo-colitis by Booker, but as they were principally confined to the submucous tissues he rightly assigned to them the role of a secondary infection.

The most important step in advance in the bacteriology of ileo-colitis was made by Duval and Bassett, who in the summer of 1902 isolated the *Shiga bacillus* from a considerable number of cases of summer diarrhoea in infants. Since their investigations the fact that the *bacilli of dysentery* are the specific organisms in the largest number of cases of inflammatory infantile diarrhoea seems to have been fairly well established. Dyspeptic diarrhoea must, of course, be excluded from this category.

The dysentery bacilli constitute a group, the most prominent members of which are the Shiga, or alkaline type, and the Harris, or acid type. The latter is the one that has been most frequently found.

The dysentery bacilli may be looked for with practical certainty where dysenteric symptoms are present, *i. e.*, fever; mucus and blood in the stool; prostration. They are not, however, confined to any one type of pathologic lesion in the gut and may even set up choleraic symptoms as in a case reported by Booker.

Among the *predisposing causes* to infectious diarrhoea *hot weather* stands pre-eminently. Indeed, so prevalent are these diarrhoeas in the hot months of the year that the popular appellation, "summer-complaint," still clings to them. It may be said to be characteristic of gastro-enteric intoxication and ileo-colitis that they rage epidemically in July and August.

*Age* is a strong predisposing cause. The majority of cases occur during the early *teething period*, *i. e.*, from the sixth to the eighteenth month. This generally brings an infant born the previous year into its *second summer*.



*Food.* The importance of food as an etiologic factor is only secondary to that of temperature and humidity. Children that are exclusively breast-fed rarely develop ileo-colitis and then probably only through lack of hygienic care. The fact that an infant is breast-fed does not exclude the possibility of its being infected if the nipples are not kept clean or if they are fissured. Again, ordinary filth and the drinking of contaminated water may be the source of infection. The vast majority of cases, however, are artificially fed without proper efforts being made at sterilization of bottles and nipples and with disregard for the source of the milk. It is always the safer plan to use pasteurized milk in the summer. Cold weather seems to give a surprising immunity to diarrhoeal affections even when the quality of the milk is none too good. The investigation of Holt and Park (*Archives of Pediatrics*, Dec., 1903) into this subject has shown the rather startling fact that despite the large number of bacteria that were found in many samples of milk fed to infants in the winter there was a remarkable tolerance for the same on the part of these infants. The practice of boiling milk and using pasteurized milk, now becoming more general among all classes, has done much toward reducing the infantile death-rate.

The *environment* is an important factor. Fresh air and personal cleanliness are two of the strongest prophylactic measures in infantile diarrhoea and when infants are kept in squalid, poorly ventilated or crowded quarters and not regularly bathed they offer poor resistance to the invasion of a serious intestinal affection. Under such circumstances the feeding is also likely to be conducted in a most careless and dirty manner.

*Intestinal indigestion* is often the first step in the clinical course of ileo-colitis and infants who are allowed to go on with such a condition, from faulty feeding and neglect, will sooner or later develop cholera infantum or ileo-colitis. Certain *constitutional* diseases predispose to enteritis. They are

notably rickets, syphilis and tuberculosis. Diarrhœa may be a terminal event in malnutrition, marasmus and tuberculosis. Certain of the *acute infectious diseases* are ushered in with diarrhœa (pneumonia, scarlet fever) or they become complicated with the same (measles, whooping cough).

#### CHOLERA INFANTUM ; ACUTE GASTRO-ENTERIC INTOXICATION.

**Infection.**—Cholera infantum is a gastro-intestinal infection of great virulence in which the symptoms are of rapid onset and of a grave character, clinically resembling Asiatic cholera.

The symptoms are the result of the absorption of toxins, which may already exist in the milk prior to its ingestion—*acute milk infection*. Vaughan has demonstrated that improperly kept milk may be sufficiently toxic to produce choleraic symptoms in animals. Pasteurization or sterilization is of no avail under these circumstances, as the toxins are not affected thereby.

Again, if unsterilized milk be fed in which the colon bacillus, or the proteus vulgaris, be present, decomposition may take place before digestion is completed and an acute *ectogenous infection* be the result.

Booker (*Archives of Pediatrics*, Nov., 1903) cites an interesting case of summer diarrhœa of the cholera infantum type in which the child was greatly prostrated and slightly toxic, the stools being watery and putrid. He looked upon the condition as one of infection with the proteus vulgaris, but investigation proved it to be practically a pure Shiga bacillus infection.

It is also possible that as a result of the enervating effects of long continued hot weather and high humidity in conjunction with improper feeding, deficient ventilation, flatulent dyspepsia (HENOCH), the micro-organisms already present in the gut, and benign under normal circumstances, may assume a virulent character and set up an *endogenous* infection.

The majority of cases are encountered during the months

of June, July and August ; this is especially so in large cities where the disease occurs epidemically. The hot weather acts both by lowering the infant's resisting powers and by interfering with the proper keeping of the food. It is, therefore, plain to be seen why the most rigid supervision of the milk supply must be enforced and why the food should be pasteurized at this time of year.

Age offers a predisposing cause ; infants are particularly susceptible during the second six months. This is a period when dentition is active and invites attacks of indigestion, besides lowering the infant's vital resistance. The importance of this etiological factor must, however, not be over-estimated.

On the other hand, cases will arise at any time of the year and in infants perfectly well to all appearances. The introduction of the poison into the system in sufficient amount is all that is necessary to precipitate the condition.

**Pathology.**—Post-mortem appearances will depend upon the duration of the disease. In rapidly fatal cases an abnormal paleness of the mucous membrane of the stomach and intestines, with slight swelling of Pyer's patches and solitary follicles, is all that is to be seen. Booker found superficial loss of epithelium of the mucosa of the stomach and gut, more or less extensive in distribution, in all fatal cases coming under his observation. The epithelium is infiltrated with leucocytes, as is also the submucous tissue. This infiltration may separate the epithelium from the underlying structure. The epithelial cells are cloudy and undergoing necrosis. The villi and follicles may be ulcerated, and, if the condition has not proven too rapidly fatal, a beginning catarrhal enteritis can be demonstrated. In gastro-intestinal intoxication the course is more protracted than in cholera infantum and under these circumstances a distinct inflammatory reaction is set up. Micro-organisms, especially streptococci, are seen in large numbers in the mucosa in cases where the superficial epithelium has been exfoliated (BOOKER).

**Symptomatology.**—Cholera infantum may begin as an at-

tack of acute indigestion, or, what is more frequently the case, suddenly, with severe vomiting and copious dejections, high fever and rapid prostration. The temperature may be high from the beginning ( $104^{\circ}$  F. and over), or it may be but slightly elevated during the entire attack. The food is promptly vomited, and later not even water or other bland substances are retained, or the vomiting may predominate over the diarrhoea. The dejections are yellowish-brown or green and fecal in the beginning, and usually painless, rapidly becoming more and more watery, until at last they consist entirely of serum. They are copious and occur from ten to fifteen times in twenty-four hours.

Collapse results from the total depletion of the system, together with the primary intoxication. With the collapse the **hydrocephaloid** state of Marshall Hall sets in, due to cerebral anæmia, with resulting venous hyperæmia and œdema of the pia mater. The child becomes somnolent and apathetic, the pulse thready or imperceptible, while the extremities are cold and cyanotic; the eyes are half closed, sunken and surrounded by dark rings; the cornea is lustreless and covered with shreds of mucus; the pupils fail to react to light, the child falls into a stupor, and death supervenes, sometimes preceded by retraction of the head and convulsions. The first symptoms mentioned can be reconciled entirely with the circulatory disturbance present in these cases, *i. e.*, fall of blood-pressure in the cerebral arteries, but when there is strabismus, retraction of the head and convulsions I am inclined to consider the condition, as viewed by Holt, Osler and other American writers, of toxic origin, especially as post-mortem findings are negative and as these symptoms are occasionally observed in other infections capable of inducing toxæmia.

The urine is scanty, frequently contains albumin and may become entirely suppressed. Necrosis of the epithelium lining the convoluted tubules is found at autopsy (BOOKER).

Owing to the great loss of fluid the child suffers intensely from thirst, and toward the end of the disease *sclerema* may

develop. This is a hardening of the skin, which begins in the feet and extends to the gluteal region, sometimes also involving the back and upper extremities, due to resorption of the fluids and fat from the skin and subcutaneous tissue, with some evidence of thickening of the same.

In severe cases a fatal issue ensues in the course of a few days; it may occur within twelve to twenty-four hours in very toxic cases. Where the heart's action can be sustained and the vomiting and purging gradually subside, a favorable termination may be looked for. The outlook, however, is always grave, cholera infantum being one of the most fatal diseases of infancy. The previous health of the child is of some value in gauging the prognosis as well as the mode of onset. A fulminating case naturally indicates a large initial dose of the poison. Breast-fed infants stand a better chance than those artificially reared.

**Diagnosis.**—The condition with which cholera infantum is most likely to be confused is ileo-colitis. From this it must be differentiated by the rapid development of the symptoms, both the primary manifestations and those developing secondarily, *i. e.*, the collapse, scanty urine and hydrocephaloid. The serous character of the stools is another diagnostic point.

During epidemics of *Cholera Asiatica* bacteriological examination of the stools would be the only positive test for differentiating the two conditions.

**Acute gastro-enteric intoxication** is a *fermental diarrhœa*, standing midway between simple diarrhœa and ileo-colitis. It is the commonest form of summer diarrhœa. The diarrhœa is set up by the multiplication of bacteria in the intestinal tract, or in the milk prior to feeding, to such an extent as to induce chemical changes. The lower fatty acids—acetic, propionic and butyric—are particularly active in this direction. Products of nitrogenous decomposition may also develop in abnormal amount and induce diarrhœa. The bacillus lactis *arogenes* is particularly concerned in the former (acid), while the proteus vulgaris and colon bacillus participate in the latter

class of diarrhoea (alkaline), although a multitude of other bacteria may produce chemical changes in the intestinal contents.

In *mild cases* the symptoms are those of intestinal indigestion. The fever, however, is higher and the stools more watery and frequent. At first they contain food particles, but later they become watery and of a dirty yellow or greenish color. The odor is exceedingly disagreeable and there is pain and flatus due to the fermentation. The stools usually pass with a loud, spluttering sound and have a frothy appearance. In the cases due to the oxidation of the carbohydrate and fat of the food the reaction is acid, while in those with proteid decomposition it is more likely to be alkaline, especially when serum is abundant. Mucus makes its appearance, but it is not as abundant as in ileo-colitis, nor is blood present.

*Severe cases* act very similar to cholera infantum. They may begin abruptly with vomiting, temperature of  $104^{\circ}$  and over, purging, nervous symptoms and great prostration. As the intestinal canal becomes emptied, however, there is an amelioration of symptoms, and under proper management the case goes on to recovery.

In a previously healthy infant the *prognosis* is more favorable than in ileo-colitis or cholera infantum.

The **diagnosis** rests upon the fact that we have here an intoxication without anatomic lesions. A protracted case, however, will set up pathological changes in the gut. From *indigestion* it is differentiated by the more severe character of the symptoms, *i. e.*, the more pronounced intoxication, the longer duration of symptoms (five to ten days) and the watery, offensive character of the stools.

From *ileo-colitis* it is to be distinguished by the absence of characteristic pathological changes in the gut. The fever is not persistent, but falls after the second or third day; there is no blood and only a small amount of mucus in the stools; pain, prostration and emaciation are less pronounced.



ACUTE ILEO-COLITIS; ACUTE INTESTINAL CATARRH;  
DYSENTERY.

The term ileo-colitis is usually adopted to designate the acute intestinal catarrhs of children, as in these cases the lower end of the ileum and the colon are more prominently affected than any other portion of the intestinal tract. Cases in which the colon and rectum are principally involved are described as *dysenteric diarrhœa*, from the distinct clinical picture they present.

This term, however, has been abandoned, as it is but a variety of ileo-colitis. Again, when the stomach is involved in the inflammatory process we may speak of *gastro-enteritis*—a term still employed by some writers.

**Pathology.**—Pathologically, we can divide ileo-colitis into *acute catarrhal ileo-colitis*; *acute membranous ileo-colitis* (*dysentery*), and *ulcerative follicular ileo-colitis*.

In mild cases the mucous membrane of the lower ileum and more or less of the entire colon appears congested and slightly swollen. In the small intestine the congestion usually appears in streaks on the folds of the mucosa which are seen to run transverse to the long axis of the gut as it is laid open for inspection. The small intestine is distended with gas and filled with undigested food and greenish mucus, which adheres to its surface. The colon is more or less empty and in cases of short duration does not show as pronounced changes as the ileum, while in protracted cases it is always more affected.

In more severe cases the deeper structures are involved, as a result of which there is slight thickening of the intestinal wall from round cell infiltration of the sub-mucosa. The lymphoid structures are also swollen from congestion and increase in the lymphoid cells and the congestion of the mucosa is more pronounced and uniform. In the small intestine there is a distinct area of congestion about Peyer's patches; the latter may stand out prominently, but they



seldom ulcerate as in typhoid fever. The lymph-follicles of the colon are the ones most markedly involved and they stand out on the mucous membrane as small beads—*follicular enteritis*. When the process has been a protracted one the follicles ulcerate. In severe catarrhal ileo-colitis ulceration, when it does occur, is more likely to take place irrespective of the lymph follicles and result in the production of variously sized, irregular, superficially situated areas (catarrhal ulceration). Hæmorrhage does not result from such ulcers but they offer a port of entry for the development of a general bacterial infection and they always tend to protract the case if they do not hasten the death of the child.

Microscopically we find destruction of the superficial epithelial layer and more or less round-cell infiltration of the mucosa. The bloodvessels are engorged and the lymphoid structures swollen. In mild cases the process stops here. In severe cases the infiltration reaches to the muscular layers and necrotic changes take place in the inflamed follicles. The epithelium is densely infiltrated with leucocytes and more or less fibrin poured out. This is but a step to the membranous variety.

*Membranous colitis* presents the most pronounced anatomic changes. It corresponds closely with dysentery as seen in adults, but the membrane is not so thick and ulceration does not occur so extensively. The membrane is practically limited to the colon, its ascending portion and the sigmoid flexure being favorite sites. The membrane is of a dirty-gray color and closely adherent to the mucous membrane, contrasting markedly with the deep red congestion of the latter where there is no membrane. The main changes are found in the intestinal wall, which is considerably thickened and rigid. Membrane may extend down as far as the rectum, where it can be seen during life as the child strains at stool.

*Follicular ulceration* is not uncommon in cases that have run a protracted course. It is especially prevalent in institutions and among poorly nourished infants that have suffered

from repeated attacks of gastro-enteric intoxication. The process is essentially a sub-acute one. Holt found it in thirty-six out of eighty-two autopsies upon infants dying of intestinal inflammation.

The appearance is characteristic. The ulcers are round, varying in size from a pin point to that of a split pea and represent destruction and excavation of the inflamed solitary follicles. They may be found in both the ileum and colon, but most frequently they are confined to the colon. While they extend as deep as the muscular layer of the gut they do not tend to perforate.

In association with the distinctive lesions of ileo-colitis we not uncommonly encounter *broncho-pneumonia* as a complication, which, in fact, may prove to be the determining cause of death in a protracted case. It is usually of the desquamative type; rarely septic, although a general infection from the intestines is possible.

In the *kidneys* we may find evidence of acute parenchymatous degeneration. Actual nephritis is rare. The *mesenteric glands* are usually enlarged.

**Symptomatology.**—A case of mild catarrhal ileo-colitis begins with fever and loose movements; intestinal fermentation; sometimes vomiting, and it cannot be distinguished from the non-inflammatory diarrhoeas until mucus and blood make their appearance in the stools. Instead of these symptoms abating after the intestinal tract has emptied itself we find rather an increment in the severity of the condition and the child continues to have small, frequent dejecta consisting in the main of mucus and a little blood. These stools are alkaline in reaction and practically odorless.

As the case advances the stools become more irregular. Some are large, containing mucus, undigested food particles and serum in abundance while again others are simply a stain of mucus on the diaper as a result of the *tenesmus* that plays so prominent a role in ileo-colitis. On account of this straining there is a strong tendency to the development of *prolapsus ani*.

The *constitutional symptoms* are fever of moderate grade, ranging between 99° F. and 102° F., although at the onset it may be much higher for a short period; prostration; loss of appetite and in some cases vomiting. The duration of these symptoms is about a week. Convalescence is slow and is characterized by a tendency to persistence or recurrence of mucus in the stools as soon as we attempt to put the child back on its customary diet.

**Severe catarrhal ileo-colitis** presents symptoms much in common with dysentery. Constitutional symptoms are pronounced. The fever is high throughout the entire course of the disease and the movements are frequent, accompanied by painful straining and consisting mainly of bloody mucus. The abdomen is not distended as a rule, but owing to tenderness it may be rigid.

Prostration and nervous symptoms are marked. The child presents the picture of a severe infection—dry, coated tongue; sordes on the teeth; apathy or great irritability; anorexia and thirst; prostration. Death from sepsis, broncho-pneumonia or exhaustion is a frequent outcome. If recovery takes place we may look for a protracted convalescence on account of catarrhal ulceration of the gut. The duration is from two to three weeks, although death may occur in the first few days.

**Follicular ulceration** is to be suspected in children of weakly constitution who have had repeated attacks of diarrhoea or a protracted moderately severe ileo-colitis and in whom mucus persists in the stools. There is also a combination of fever of moderate range. The accompanying symptoms are progressive emaciation and failure of strength; anorexia; bed sores; thrush, etc. The duration is long; the condition is practically a sub-acute one. The course is marked by improvement and exacerbation and so may be protracted for two or three months. Even after the ulcers have healed there is more or less persistent indigestion and tendency to diarrhoea for some time. The characteristic symptoms may be summed up as continued loose movements, four to eight daily, consist-

ing chiefly of greenish mucus; slight fever; emaciation; absence of blood in the stools. It is also to be remembered that follicular ulceration is the result of extension of the inflammatory process into the deeper layers of the mucosa as a result of repeated attacks of intestinal infection. It is, therefore, almost always encountered in delicate or poorly cared for infants during the teething period.

**Membranous Colitis.**—Strange as it may seem, this severe form of enteritis often presents the most uncharacteristic symptoms; indeed, we may be deceived into looking upon the condition as an entirely different disease.

When the onset is abrupt and accompanied by cerebral symptoms it closely simulates meningitis. High fever; convulsions; retraction of the head and abdomen; vomiting and stupor may be present for several days before our attention will be directed to the intestines by the appearance of bloody stools and possibly prolapsus ani.

The majority of cases, however, simulate severe catarrhal ileo-colitis with the exception that shreds of membrane appear in the stools and may be seen at times upon the rectal mucosa during prolapsus. In all doubtful cases the stools should be carefully washed and strained, as it is difficult to distinguish membrane from mucus when the latter is abundant.

The duration is from one to three weeks. It is very fatal, especially in young infants; in older children its protracted course may lead one to suspect typhoid fever on account of the continued high pyrexia.

The *diagnosis* rests upon the evidence of severe inflammation of the large intestine, especially of the descending portion. Together with continued high fever and prostration there are frequent, small stools consisting mainly of blood and mucus and there may be shreds of membrane. There is tenderness along the entire course of the colon, but particularly along its descending portion. In *typhoid fever* tenderness is only found in the ileo-cæcal region and the stools are large, consisting mainly of the contents of the small intestine.

In *meningitis* the bowels are constipated and the cerebral symptoms progress in regular order from day to day. In dysentery they are purely toxic and therefore vary ; in fact, they may clear up, while the intestinal symptoms increase in severity.

Pain, tenesmus, vomiting and prostration may suggest *intussusception*, but in this condition the onset is abrupt, there is no fever, and an abdominal tumor may be made out.

**Treatment.**—Although the treatment of the infectious diarrhoeas must have much in common with the entire group, still individual cases will require remedies and adjuvants peculiarly adapted to their characteristic symptoms. *Prophylaxis* is of the greatest importance, and is equally urgent for all cases. In the first place, the exciting cause (micro-organisms) must be most rigorously combatted ; in no case should a child be fed on contaminated food, or allowed to nurse from bottles or nipples not aseptically clean.

All discharges should be disinfected, as not infrequently diarrhoea becomes epidemic in a family or hospital ward.

The supervision of the food is of the greatest importance. Use only pure, clean milk. Boil the water you give the babe. Pasteurization will not make dirty milk wholesome. If chemical changes have occurred in the milk, sterilization will not prevent it from acting as a poison. Another important point in prophylaxis is not to wean an infant during the summer. There are times when this becomes necessary, but whenever at all possible we should wait for the advent of cool weather before taking this step.

Prophylaxis, therefore, resolves itself into regulating the child's surroundings and most rigorously attending to every detail of feeding. In summer no infant should be kept in the city if the parents can afford to take it away. The country is good ; the seashore is better. Even after the infant is seized with ileo-colitis it is not too late to take it out of the city, and its recovery may depend upon this step.

The poor and overcrowded are particularly unfortunate and

for them we have not sufficient charities to give them the cheapest of nature's offerings,—fresh air. Let them keep the children indoors all day in the coolest room, with the shutters closed in to keep out the broiling sunlight, and after sundown and early in the morning take them out to the neighboring squares and parks for an airing. Trolley rides and trips on the river are fortunately cheap and will help to save many a baby's life.

Bathing is most essential during hot weather. The cool or tepid bath is absolutely necessary when fever is present, and it may be given three to four times a day. Chapin recommends allowing the children to play in a bath tub partly filled with luke-warm water.

Even though we may be able to obtain milk that has been handled in the most careful, up-to-date manner, and we know it to be clean and reasonably free from micro-organisms, still I believe we are taking chances if we do not pasteurize it in hot weather. An error in the technique in the preparation of the infant's bottles at any one point of the process may result in a fatal case of ileo-colitis.

The nipples should be boiled every day and the bottles filled with hot water and washing soda as soon as emptied. Then before refilling they should be cleansed with a bottle-brush and thoroughly rinsed with hot water.

When maternal feeding is practiced the nipples should be washed before and after nursing with a saturated solution of boric acid. The infant should not nurse directly from a fissured nipple. Here it is better to employ a sterilized shield or pump out the milk and feed it with a spoon. These methods are preferable to attempting to cleanse an infant's mouth after nursing.

In hot weather infants get thirsty between feeding times and should receive an ounce or two of water, previously boiled and then cooled.

A most important point to bear in mind is that during hot weather an infant cannot, as a rule, take the same strength of



fat and proteids it is able to digest at other times. It will usually take the same quantity because it is thirsty, but unless we cut down percentages we may set up a severe indigestion which in turn will invite enteritis. Do not expect a babe to make its regular weekly gain in weight during July and August.

When diarrhœa has developed we must at once make material changes in the feeding. In a breast-fed infant, in the absence of fever and vomiting, we may for a day or two continue with the breast milk and wait for the action of our remedies. Should the condition not improve it will be well to alternate a bottle of barley-water with the breast and in that way give the digestive organs a rest. Should the condition get progressively worse in spite of this we must stop the breast entirely.

The reason milk is discontinued in diarrhœa of infants is because it acts as a good culture medium for the micro-organisms that are causing the trouble, and the curds of casein act as an irritant to the mucous membrane. In acute ileo-colitis milk, even if sterilized, is practically a poison.

Barley-water as an all-round substitute food is, perhaps, the most generally useful one. It leaves very little residue in the gut and starves out the bacteria. Sometimes it disagrees or is objected to by the infant. I have previously pointed out that the particles of cellulose found in barley-water may irritate the inflamed mucous membrane. In such cases I use arrowroot, which is more bland and more acceptable to some infants. In protracted cases the infant will lose too much flesh if we only give it barley-water, and as milk may have to be withheld for several weeks in isolated cases, particularly when there is follicular ulceration, we must use a more nourishing milk substitute. Here I like to alternate lamb-broth made with rice and then strained, and barley-water containing half-ounce of sugar of milk and the white of one egg to the pint. The twice-boiled flour-ball is another excellent food, especially for very young infants. A return to milk must



be made cautiously, beginning with low percentages, especially of proteids. A tablespoonful of milk to four ounces barley-water is a safe beginning. Sometimes it will be found beneficial to restrict cases with alkaline stools exclusively to carbohydrates and those with acid stools to broths.

*Special Symptoms and Their Management.*—*Vomiting* is at times a most troublesome complication, especially in cholera infantum. Lavage of the stomach is the most rational and successful method of treatment to control it. In urgent cases it may be necessary to perform the operation several times a day, and then pour a little food into the stomach before removing the tube. Thin arrowroot-water or albumin-water is best retained under these circumstances.

Often the food will be retained better if fed with a teaspoon than when taken from a bottle. When the infant can take only a small quantity of food at a time we must feed it often, but there is no good in feeding every five or ten minutes, as is sometimes done.

*Diarrhoea.*—In the early stages of an intestinal infection we will derive much benefit from bowel irrigation. It is rare that the gut thoroughly empties itself at once, and if the abnormal intestinal contents are allowed to remain undisturbed for any length of time inflammatory changes in the intestinal mucosa will result. It is true, the irrigating fluid does not reach beyond the ileo-cæcal valve, but, as the colon receives the brunt of the attack in most instances, we help the case materially by cleansing this part of the gut. Besides, irrigation stimulates peristalsis, and thus aids in emptying the portion of gut above this point.

We often encounter a condition of high fever with frequent, small stools, consisting chiefly of mucus and a little blood. The abdomen is distended with gas and the gut laden with decomposing fecal matter. Here irrigation is imperative. In such cases mild, cautious purgation is justifiable, but I must warn against the indiscriminate and injudicious use of the initial *Calomel* purge. I can recall several cases in which every

chance of recovery was spoiled by the superadded irritation induced by *Calomel*.

Persistence of mucus in the stools calls for irrigation, but we must stop this procedure as soon as the bowel begins to empty itself naturally. Many a diarrhoea is kept up by too much mechanical interference.

When tenesmus is persistent we can give the child much relief by injecting a small amount of warm olive oil into the rectum. This exerts a soothing influence upon the inflamed membrane.

*High fever* is best controlled with the bath. Infants may be tubbed two or three times daily in water gradually reduced from 90° F. to 80° F., while older children are more conveniently sponged with cold water and alcohol. Irrigation also tends to control the pyrexia. The child should be kept in the open air as much as possible.

*Collapse* requires active stimulation. Brandy should only be used when called for, and not given continually during the illness. In grave cases a hypodermic injection of camphorated oil may be necessary. Five minims may be given to an infant one year old. *Camphor* suits this condition admirably, and it is best given hypodermically, as it may otherwise irritate the stomach. Most cases of cholera infantum will need it sooner or later. Artificial heat must be applied also when the body surface becomes cold or the temperature subnormal. I have at times seen beneficial results from hypodermoclysis, injecting an ounce or more of normal saline solution into the abdominal subcutaneous tissue with an antitoxin syringe. These cases are so grave, however, that often nothing will do the slightest good.

*Remedies.*—While each case should be individually prescribed for, still we can more or less successfully classify our remedies in accordance with their applicability to the different varieties of infantile diarrhoea.

In simple intestinal indigestion *Nux vomica* is most useful. When given in time it will often cut short an attack. Hughes

recommends *Lycopodium* when the condition becomes inflammatory. Teste speaks of this remedy as a specific in infantile enteritis.

Some infants are predisposed to diarrhœa without any apparent cause. In these cases there seems to be a slight catarrh, such as we find in the respiratory tract. *Pulsatilla* is very valuable here. The diarrhœa accompanying teething is especially benefited by *Chamomilla*. In acute gastro-intestinal intoxication *Belladonna* appears most frequently indicated on account of the predominance of fever and nervous symptoms. Even in the later stages, when the bowel symptoms become more prominent, I have found *Belladonna* invaluable as long as fever and toxæmia were present.

In the ordinary case of fermental diarrhœa and ileo-colitis I find *Podophyllin* 2x trit. a good routine remedy. *Mercurius vivus* 3x trit. follows, if ulceration takes place. This is indicated by the continuance of the diarrhœa, moderate fever and abundant mucus in the stools. In the dysenteric type of colitis, *Mercurius corrosivus* 6x is the chief remedy.

*Arsenicum*, *Ipecac* and *Veratrum album* are the most useful remedies in cholera infantum. *Veratrum* is Jousset's favorite. *Iris versicolor* will check the vomiting speedily, but leaves the bowels untouched according to Richard Hughes. *Arsenic* and *Veratrum* are often difficult to differentiate, especially in the beginning of the case. Under these circumstances there is no objection to alternating. I have often found that when one of the apparently indicated remedies failed to act, prompt improvement followed on giving a constitutional remedy in alternation. Among these *Calc. phos.* stands foremost.

Goodno cites a remarkable result obtained from the use of *Zincum* 6x trit. in a case of collapse with abolition of all reflex excitability, together with a cessation of vomiting and diarrhœa. For a more detailed study of the therapeutics of diarrhœa the following remedies are appended:

*Acon.*—In the beginning; high fever and restlessness; green mucus in the stools.

*Æthusa*.—Vomiting of large curds, followed by prostration; projectile vomiting; convulsions.

*Antimon. crud.*—Tongue heavily coated white; disposition much changed, making the child disagreeable and fretful.

*Apis*.—Cerebral symptoms; suppression of urine; coma, with hot head, dry skin; shrill cry.

*Arsen.*—Watery stools, with vomiting and collapse; stools offensive, first greenish, later becoming dark or brownish, acrid; also small mucus stools with tenesmus. Child nurses often, but takes only a small quantity at one time. Mainly differentiated from *Veratrum album* by concomitant symptoms, although where *Arsenic* is indicated pathological changes have already occurred in the bowels, blood and viscera. The symptoms are violent from the beginning, or, if less acute, are marked by a progressive downward tendency.

*Bell.*—Green stools, abdomen distended and sensitive; face red, high fever. Where inflammatory symptoms are pronounced *Belladonna* is the most important remedy, especially when brain symptoms make their appearance.

*Bry.*—Brought on by change of weather; stools brownish, worse from motion; great thirst for large quantities of water.

*Calc. carb.*—Stools light in color, sour odor; sour vomiting; dentition; rachitic tendency; belly large.

*Calc. phos.*—Child looks old, under-developed; stools greenish, thin and offensive; history of tardy dentition; belly flabby. A most valuable tonic both during the disease and in convalescence.

*Carbolic ac.*—When the vomiting is a distressing feature, two or three drops in half a glass of water, half teaspoonful every half hour.—(CHAS. D. CRANK.)

*Camphor.*—Sudden appearance of choleraic symptoms; great prostration and collapse; body cold, apathetic state; will not remain covered.

*Cham.*—Stools green, with white particles, looking like "spinach and chopped eggs;" fretful; one cheek red, the other pale; child wants to be carried about.

*China*.—Undigested stools; movements watery and yellow; much distension of abdomen and colicky pains. *China* acts as a tonic in protracted cases.

*Colocynth*.—Painful cases; pressure gives relief.

*Croton tiglium* is a remedy I have frequently used with marked success in gastro-enteric infection where the stools are profuse and watery and of a yellow color. The mother will also tell you that every time the child takes its bottle it has one of these movements, drinking apparently exciting peristalsis and bringing on a stool. It is distinguished from *Podophyllum* by a less amount of gas and mucus and absence of straining.

*Cupr. ars*.—Painful cases; choleraic and convulsive symptoms predominate.

*Ferrum phos*.—Dr. E. L. Clark, of Media, Pa., has called my attention to the value of *Ferrum phos*. in the early stage of ileo-colitis when there is high fever and blood-streaked mucus in the stool.

*Ignatia*.—Prolapsus ani; cerebral symptoms developing suddenly.

*Ipecac*.—Nausea and vomiting; stools green as grass, or like yeast. Early stages of cholera infantum.

*Iris*.—This remedy has yielded excellent results in cholera infantum, but is also useful for other diarrhœas accompanied by vomiting. The vomited matter is sour, the dejections are thin and tinged with bile.

*Mag. carb*.—Sour diarrhœa and vomiting; stools green, like frog-pond scum (fermentation).

*Mag. sulph*. — Dr. Frank H. Pritchard (*Hahnemannian Monthly*, Nov., 1900) reports favorable results from the use of a weak solution of the *Sulphate of Magnesia* in the summer diarrhœas of children. His dosage is one-half to one grain dissolved in a teaspoonful of water. The indications calling for it are copious, watery stools, deficient in bile. He noted that as soon as the remedy had begun to act favorably the stools became bile-tinged. *Podophyllin* has a similar action.

*Mercurius*.—A predominance of mucus and involvement of the rectum calls for *Mercury*. The *Bichloride* is often preferable to the metal in dysentery; *Calomel* has grass-green stools. The “never-get-done” feeling of *Merc. sol.* is very characteristic, while the *Bichloride* has tenesmus of the bladder as well as rectum, and is the chief remedy in membranous colitis.

*Podophyllum*.—Painless, yellowish or greenish, water diarrhoea; prolapsus ani. As a routine remedy, I must acknowledge *Podophyllin* 2x trit. as the one most generally useful. When the stools are thin and greenish, expelled with considerable gas—an indication of fermentation—this remedy should be pushed until the normal yellow color reappears and the consistency is changed to a more pasty character.

*Sulphur*.—Excoriating stools, worse mornings; marantic cases. The child is peevish and has a voracious appetite. The lips are very red and the anus become excoriated. • Unhealthy condition of the skin. “It is especially useful in *dysentery* after *Aconite* has removed the acute symptoms, when the tenesmus has ceased but blood is still discharged.” (Bell).

*Veratr. alb.*—Vomiting and purging, the latter most prominent; motion aggravates all symptoms; cold sweat on forehead. There is less prostration and thirst than under *Arsenicum*, less restlessness and usually more pain, and when any doubt exists as to a choice between the two, *Veratrum* should receive the preference early in the case. When *Arsenicum* becomes indicated the patient has passed into a state of profound exhaustion, from which it is difficult to recall him.

**Dysentery.**—ALOES, *Apis*, ARS., *Baptisia*, *Bell.*, CANTHARIS, *Capsic.*, *Colch.*, IPECAC., *Kali bichr.*, *Lach.*, MERC., MERC. CORR., *Nux vom.*, RHUS TOX.

**Hydrocephaloid.**—*Æthusa*, APIS, ARS., *Bell.*, *Borax*, *Bry.*, CAMPH., *China*, CUPR., HELLEB., *Ignatia*, *Veratr. alb.*, ZINC.

#### AMŒBIC DYSENTERY.

Normally amœbæ are never found in the stools of children; rarely monads are encountered. Amœbic dysentery is uncom-

mon in children, but the fact that Amberg (*Johns Hopkins Hosp. Bull.*, Dec., 1901) met with five cases in his clinic (Baltimore) in a single winter indicates that the disease at least merits attention. Amberg's contribution on this subject is not only most thorough and comprehensive, but at the same time brings out most valuable data upon the state of the blood.

The disease is contracted by drinking contaminated water. Well-water may be such a source, or children will drink water from the gutter, as in two of Amberg's cases.

The course is chronic and usually of moderate intensity (COUNCILMAN). The general health is not much disturbed, although the child becomes anæmic. The main symptoms are intestinal, *i. e.*, pain and discomfort in the bowels; two to five loose stools daily with tenesmus, the stools containing mucus and more or less blood; slight fever, not necessarily continued. Complications are rare. Abscess of the liver, however, has been encountered.

In the stools motile amœba, characteristically containing red blood-corpuscles, are found.

Anæmia, more due to a deficiency in hæmoglobin than to a reduction in the number of red corpuscles, results. Leucocytosis is present. The polymorphonuclear neutrophiles and the eosinophiles are increased. When the anæmia becomes pronounced megaloblasts and microcytes; polychromatophilic erythrocytes and poikilocytosis may be encountered. Myelocytes were also seen in severe cases (AMBERG).

#### CHRONIC DIARRHŒA; CHRONIC GASTRO-INTESTINAL CATARRH OR MUCOUS DISEASE.

Chronic intestinal catarrh may result from an acute attack, or it may be the outcome of a constitutional dyscrasia, such as scrofula, rickets, tuberculosis, anæmia, etc. Some of the infectious fevers are very prone to be followed by a more or less chronic diarrhœa, notably measles and whooping-cough, especially the latter, after which the so-called *mucous disease*



frequently sets in. A physiological predisposition to excessive secretion of mucus from the alimentary tract belongs to childhood. There is no evidence that simple chronic catarrh of the gut is necessarily of bacterial origin. No doubt a disturbance in the innervation of the secretory glands underlies a large number of cases, and it is reasonable to suppose that the debilitating influence of an infectious disease may readily cause such a disturbance.

**Pathology.**—The pathological process is not confined to any particular locality in the intestinal tract, and may affect both the large and small intestines with equal severity. The mucous membrane presents a grayish appearance, with areas of injection. The solitary follicles are enlarged, and usually ulcerated. This results from secondary infection. Mucus is present in abundance, and infiltration of the submucosa with leucocytes, together with dilatation of the capillaries and compression of the glands of Lieberkuhn, are seen microscopically. The glands gradually atrophy, and in the late stages the mucous membrane presents a wasted appearance.

**Symptoms.**—Frequent, foul, loose bowel movements, together with progressive emaciation, anæmia, later œdema of the extremities, or general anasarca and death, are the result of severe intestinal catarrh, with atrophic changes. In tuberculous ulceration hæmorrhages are likely to occur, and the stools contain tubercle bacilli and round cells.

In milder cases the symptoms are not so persistent, and will depend upon the location of the process. The distinction is not so marked here as in acute diarrhœas, but we can often decide whether the small or large intestine is most prominently affected; especially is this the case in the so-called *chronic dysentery* (see *Amœbic Dysentery*).

In older children there is, beside the diarrhœa, distention of the abdomen, coated tongue, offensive breath, dark rings under the eyes, gritting of the teeth at night, and many other symptoms suggestive of “worms.” Often constipation alternates with diarrhœa. As intestinal parasites will set up a

catarrhal condition of the gut it is plain to be seen why similar symptoms are present in both conditions.

A chronic diarrhoea during infancy is frequently the forerunner of rickets; in older children it predisposes to tuberculosis.

In mucous diseases the entire alimentary tract is involved. The tongue is anæmic, flabby, and glossy in appearance. It may be fissured or mapped. The appetite is impaired and the breath is offensive. Naturally the child's nutrition is impaired from the mechanical interference with the process of digestion and assimilation caused by the mucous secretion and the child looks pale and emaciated.

The *mucous flux*, as Eustace Smith calls it, usually occurs paroxysmally and may be preceded by premonitory symptoms, such as chilliness and abdominal discomfort. There may be a large number of mucous stools a day or only one; indeed, constipation may alternate with the discharge of a large quantity of accumulated mucus.

The child is neurasthenic; often afflicted with night terrors or enuresis, and a variety of other nervous disturbances may be found associated.

The *course* is prolonged and tedious. Some cases improve temporarily and then relapse; others persist during life to some extent.

**Diagnosis.**—The diagnosis of a chronic diarrhoea becomes self evident, but the differentiation of tuberculosis of the intestines from simple catarrh is often impossible without a microscopical examination of the stool. The discovery of tuberculous disease in the lungs or elsewhere, together with the presence of enlarged mesenteric glands or chronic peritonitis and hæmorrhages, is strong evidence of tuberculous ulceration of the gut.

In simple catarrh we should determine whether it be primary or secondary to some constitutional or organic disease, such as rickets, malaria, nephritis. Mucous disease is readily recognized by the character of the stools.

**Treatment.**—The diet should consist largely of milk, which may be given pure or modified, according to the age of the child. Meat should be avoided. Plenty of fresh air or a trip to the country are of decided benefit. Irrigation of the bowels may be employed when the stools are very offensive.

Children that are rapidly losing weight and strength require mild stimulation.

In severe cases of mucous disease the diet must be most rigorously supervised. It is often advisable to give nothing but water for several days ; then cautiously adding beef juice ; poached eggs ; dry toast or zweibach ; rice ; skimmed milk ; egg-nog.

Lavage and irrigation of the colon are most valuable adjuvants in the treatment of mucous disease.

*Arg. nitr.*—Stools worse at night, or immediately after eating ; craving for sweets.

*Ars.*—Worse at midnight ; stools brownish, very offensive, excoriating ; senile appearance.

*Calc. c.*—Stools light or sour ; scrofulous diathesis ; pot-belly ; sweating about head ; mesenteric glands enlarged.

*Calc. phos.*—Abdomen flabby ; stools greenish ; dark-complexioned, puny, undeveloped children.

*China.*—Diarrhœa, with much flatulence ; anæmia, prostration.

*Graph.*—Abundant whitish mucus in stools.

*Hepar.*—Scrofulous subjects ; skin eruptions, acid dyspepsia, craving for sour things ; fat-diarrhœa.

*Ipecac.*—Clean tongue, nausea, constant pain in umbilical region, malarial type.—(W. L. DODGE.)

*Lach.*—Stools very offensive ; croupous enteritis ; great sensitiveness in ileo-cæcal region.

*Kreosotum.*—Stools grayish, very offensive ; child belches a great deal when carried about ; old-looking children.

*Merc.*—Stool contains mucus in abundance, is excoriating, greenish, and voided with much straining.

*Phos.*—Painless watery diarrhœa. The anus is relaxed and open.

*Phos. ac.*—Painless yellowish diarrhoea, with great rumbling in the intestines.

*Sulphur.*—Worse early in the morning; variable in color, excoriating; prolapsus ani; child is greedy; mouth and lips dry and very red; old expression; the skin is dry or eczematous in various localities; aversion to being washed.

**Mucous Disease.**—The chief remedies in this affection are the *Mercuries*; *Kali bichromicum*; *Colchicum*; *Calcareo phosphorica*; *Lycopodium* and *Argentum nitricum*.

*Merc. viv.*—This remedy I always prefer in any case of diarrhoea with an excessive amount of mucus. Should it seem insufficiently active the *Bichloride*, 3rd decimal trituration, cautiously administered, should be tried.

*Kali bichrom.*—Best suited to cases with pronounced gastric involvement. Dry, red, smooth and cracked tongue; nausea and vomiting; craving for acids; gelatinous stools.

*Colchicum.*—In the early stages. Stools consist of jelly like mucus; sensation of coldness in stomach; tympanites. Gripping before stool.

*Calc. phos.*—Many cases will require *Calc. phos.* as a constitutional remedy. The same may be said of *Lycopodium*, which is indicated more on its general symptoms than on the character of the stool.

*Arg. nitr.* 3x trit.—Stools consist of green mucus mixed with undigested food particles (*Calc. phos.*) and are usually expelled with considerable gas. The face is pale, sunken, and old looking. Mouth dry; tongue cracked, with viscid mucus; aphthous sores in mouth. Nervous symptoms are prominent (*Lyc.*). Craving for sweets.

#### INTESTINAL TUBERCULOSIS.

Primary intestinal tuberculosis is by no means as common a condition as was at one time supposed, and while quite a number of authentic cases are on record, still the development of tuberculosis primarily in the intestinal tract is rather the exception than the rule. Secondarily, tuberculous ulcera-

tion of the intestines is encountered in from one-third to one-half of all tuberculous cases coming to autopsy, being less frequent in infants than in older children, no doubt owing to the fact that infants succumb to the disease before the intestines have had an opportunity of becoming infected ; also because of the different course assumed by tuberculosis in infancy, there being rarely a breaking down of pulmonary tissue with the formation of bacilli-laden sputum.

Again, the belief that feeding infants upon milk from tuberculous cows is responsible for the majority of cases of infantile tuberculosis has been largely abandoned. It is conceded that bovine tuberculosis may, under circumstances most favorable to its transmission, be implanted upon the human organism and set up a primary intestinal tuberculosis, but this rarely takes place. Koch, in his famous address before the British Tuberculosis Congress (July, 1901) attempted to prove that bovine tuberculosis was distinct from human tuberculosis and was never transmitted, but in this he is not unanimously supported. The requirements for infection by milk seem to be that the milk come from a cow suffering with tuberculosis of the udder, that the milk contain a great number of bacilli and be ingested in large quantities and practically constituting the sole diet of the subject in question. This being the case with infants and young children they are the ones mainly exposed to danger (NOCARD).

**Pathology.**—The favorite seat of the lesions is the small intestine, near the ileo-cæcal region. Sometimes ulcers are found in the cæcum, but rarely in the colon. Associated with ulceration there is almost invariably infiltration of the mesenteric lymphatic glands, and when the condition has existed for some time they become caseous. In some instances the affection of the lymph nodes plays the most prominent role. They become prominently enlarged, making it possible to feel them through the abdominal wall, and on account of the interference with the process of assimilation emaciation becomes most pronounced. This represents the so-called *tabes mesenterica*.

In the early stages of intestinal tuberculosis small, yellowish nodules representing infiltration of the solitary follicles and Peyer's patches are encountered. They break down, leaving an ulcerating surface, with irregular border and overhanging edges. The characteristic features of the tuberculous ulcer are its outline and border, and the direction of its long axis, which is at right angles to that of the gut. This is directly opposite to the direction assumed by typhoid ulcers, which run in the same direction as the gut. At times a tuberculous ulcer is encountered, almost completely encircling the gut. As the process is a slow one, the surface of the ulcer is covered with granulations, and perforation rarely occurs, owing to localized plastic inflammation of the serosa accompanying the process.

**Symptomatology.**—Chronic diarrhœa with the occasional appearance of blood in the stools, or at times a severe hæmorrhage, are the characteristic symptoms of tuberculous ulceration of the intestine, especially when these symptoms appear in a tuberculous subject. Diarrhœa is, however, not always present, and hæmorrhage may be absent to the last. Again, diarrhœa is frequently associated with tuberculosis without any evidence of ulceration being present. Cases presenting the greatest difficulty in diagnosis are those of primary origin.

The data upon which a diagnosis is to be based are the following: Persistent diarrhœa, with occasional bloody stools or a hæmorrhage in a child presenting evidence of tuberculous lesions elsewhere; enlargement of the mesenteric lymph nodes; tenderness in the ileo-cæcal region; emaciation; the presence of tubercle bacilli in the stool; gradual onset of the symptoms.

The *prognosis* is unfavorable. Diarrhœa is always an unfavorable event in a case of tuberculosis, as it interferes with the proper feeding of the patient—his most important weapon against the inroads of the disease. Primary cases, or such in which the disease has not made prominent headway elsewhere, offer the best prognosis.

**Treatment.**—In the presence of diarrhœa the food must be carefully selected, avoiding meat and indigestible solid food. Soups, milk, raw eggs, crackers and dry toast are the main articles of diet that are suitable. In case of a hæmorrhage, food is best withheld for a time and an ice-bag placed on the abdomen. Supra-renal extract may be administered, one to two grains hourly, for several hours.

The following remedies should be studied :

*Arsenicum jod.*, 3x trit.—A series of cases of abdominal tuberculosis in which diarrhœa was a prominent symptom has been reported by Day (*Monthly Hom. Review*, No. 10, 1897). He claims to have obtained excellent results from *Ars. jod.* in these cases. Personally, I prefer *Iodoform*, unless there be pronounced indications for *Arsenic*.

*Calc. carb.*—Pot-bellied, emaciated children ; tabes mesenterica ; scrofulous diathesis ; pale or grayish, offensive stools.

*Calc. phos.*—In *Calc. phos.* diarrhœa is the most prominent symptom, while in *Calc. carb.* the affection of the mesenteric glands plays the most prominent role. The child is emaciated ; abdomen soft and flabby ; stools frequent, undigested and mixed with greenish mucus.

*Iodoform* 3x trit. has given most excellent results in chronic diarrhœa with emaciation, distention of the abdomen and enlargement of the mesenteric glands. It is not unusual to find evidence of tuberculosis elsewhere, such as infiltration of the lungs and tuberculous adenitis. As a rule, the appetite soon improves under its administration and the character of the bowel movements changes decidedly for the better.

*Kreosotum.*—Offensive, grayish stools ; old looking, emaciated children. *Kreosote* is a valuable remedy in the diarrhœa of syphilitic infants.

*Nitric acid.*—Ulceration of the bowel, with bloody stools ; cracking of the corner of the mouth ; ulceration of the mouth and tongue ; strong smelling urine.

*Phosphoric acid* is a valuable remedy in the debilitating diarrhœas secondary to pulmonary tuberculosis.



## CONSTIPATION.

Constipation is common during infancy, and is especially likely to result from the use of an unsuitable food and the neglect of certain hygienic measures. In regard to the former, insufficient fat and excess of proteids or starchy food, or the exclusive use of boiled milk, is a common error, and irregularity in feeding; insufficient exercise, bathing and fresh air are additional causes. Constipation in infants sometimes results from insufficient food or a milk formula that is too weak. Owing to the great length of the intestinal tract and the exaggerated curve of the sigmoid flexure (JAKOBI), together with the feeble muscular coats of the gut, fæcal residue in either insufficient or excessive amounts is usually expelled with difficulty or retained until its moisture is absorbed. Congenital dilatation and hypertrophy of the colon (HENOCH) has also been found, together with obstinate and fatal constipation.

Physiological disturbances inducing constipation are insufficiency of biliary and intestinal secretion.

Later in life we encounter constipation depending upon errors in diet, irregularity in attending to the calls of nature, insufficient fresh air and sunshine, and intestinal indigestion, but we must be wide awake to the fact that frequently it is but a symptom of some constitutional ailment of far greater importance, namely, rickets, tuberculous meningitis, anæmia and various organic disturbances.

Painful local affections often lead to constipation through a dread of evacuating the bowels. In such cases I have seen impaction of fæces with obstruction of the bowels result, making it necessary in one case to incise the sphincter in order to remove the hard scybali.

**Symptoms.**—As constipation is but a symptom in itself we must search for the cause, remedy that, if possible, and apply the necessary measures toward the relief of the intestinal torpor. Although the stool is usually hard, dry, lighter in color

than normal, and passed in small pieces, still this is not invariably the case, for frequently the stool is soft, papescent, and of the proper quantity and color, the rectum seemingly being unable to expel it. I recall the case of a child five years old complaining of intense pains in the sacral region simulating sacro-iliac disease. The bowels moved daily, but scantily and with pain. An examination of the rectum under an anæsthetic revealed a complete impaction of the rectum and sigmoid flexure with hardened fæces, which was with difficulty removed.

At times we are able to feel masses of impacted fæcal matter in the colon by abdominal palpation. This may be mistaken for a tumor, but its characteristic putty-like consistency and its tendency to move along the intestinal tract are sufficient data to eliminate errors of this kind. A routine examination of the abdomen for fæcal concretions should be made in all cases where such a condition is likely to occur. The physician who follows this rule will be surprised to find how often, especially in acute diseases, an impacted colon and sigmoid flexure will be encountered. I would also advise a more frequent resort to digital rectal examinations.

**Treatment.**—Infants fed on artificial food should have the percentage of fat, and sometimes the sugar, increased or a predigested carbo-hydrate added to the milk, such as Liebig's Food or Mellin's Food. Breast-fed infants often receive much benefit from an occasional teaspoonful of olive oil. A drink of sugar-water once or twice daily will often act beneficially.

In the case of older children the same measures hold good, but as their diet is more varied and more easily regulated, dietetic treatment is more satisfactory here. A little honey at breakfast, together with oatmeal, graham wafers and fruit, and at the subsequent meals the addition of stewed fruits, green vegetables and the avoidance of meat and starchy foods in excess, yield most satisfactory results. At the same time the child must be encouraged in the free use of water be-

tween meals and regular habits at stool. A teaspoonful of olive oil after meals is very beneficial.

*Enemata* are of decided value in habitual constipation, and are necessary to soften the stool where anal fissures exist. If the enema be not given in excessive quantities, and nothing more irritating than soap and water or normal saline be used, there is no danger of creating an enema habit.

*Massage* is also a most valuable adjuvant to the therapeutics of constipation, and is more applicable to infants than to older children. (See chapter on "Nursing.")

The most useful and most frequently indicated remedies for the uncomplicated cases are *Alumina*, *Bryonia*, *Nux vom.* and *Sulphur*.

*Alumina*.—The stool is soft and papescent; child makes no effort to move its bowels, and if so, they are usually unsuccessful; stool sticks to the anus like putty.

*Bryonia*.—Stools large and dry, as if burnt. "Constipation after castor oil."

*Graphites*.—Stool consists of small balls bound together by mucus; fissura ani; eczema ani; fat babies with skin eruptions.

*Lycopodium*.—Flatulent distension of abdomen; red sediment in urine; child cries when attempting to pass the stool owing to painful contraction of the sphincter.

*Mercurius dulcis* 2x trit., a tablet three times a day for infants, or two tablets four times daily in older children, is indicated when the biliary and intestinal secretion is deficient. This remedy should not be given over a prolonged period of time.

*Ox-gall* is a harmless substance possessed of decided chologogue properties and is a valuable remedy in many cases of constipation. It may be given in one-grain chocolate-coated tablets.

*Nux vomica*.—The child strains and grunts, but passes little or no stool; the abdomen is distended, herniæ are apt to protrude from the constant straining and kicking.

*Plumbum*.—"This remedy is suited to infantile constipation where the moisture of the stool has been absorbed and it is hard and lumpy, causing fissures, and is voided with difficulty, requiring severe straining."—(FISCHER.)

*Sulphur*.—Constant urging with prolapsus ani or hæmorrhoids, which bleed profusely at times. Habitual constipation; infantile atrophy and malnutrition; anus very sore after stool; intestinal indigestion, lips red, tongue dry and papillæ prominent through a dirty coating; hunger between meals; urine offensive with greasy pellicle; dry, unhealthy skin.

Beside these remedies, one of the following may suggest itself from its prominent objective local and general symptoms.

*Ammonium mur.*—Hard, crumbling stool, followed by a soft stool, covered with a glairy mucus.

*Calcareæ carb.*—Stools large, light; oozing of offensive fluid from anus. \ Rickets.

*Caust.*—Much urging and straining at stool, with redness of face and anxiety; passed best in the standing position.

*Chelid.*—Stool like sheep's dung; liver sensitive.

*China.*—Light stools; difficult, even when soft (*Alumina*).

*Ferrum.*—Anæmia; flushing of face.

*Hydrastis.*—"After purgative medicines."—(GOODNO.)

*Kali carb.*—Stool too large to be expelled; proctalgia.

*Nat. mur.*—Stools hard and dry, producing fissures of anus.

*Nitr. ac.*—Stool hard and scanty; fissures with splinter-like pains.

*Opium.*—Obstinate constipation; stool consists of small, hard, black balls.

*Phos.*—Stool long and narrow.

#### ACUTE INTESTINAL OBSTRUCTION.

Obstruction of the bowels may result from a variety of causes, the most frequent being *intussusception*. The obstruction occurring in *appendicitis* is in reality a paralysis of the bowels due to septic peritonitis.

A twisting of the gut, known as *volvulus*, is occasionally met with in children, who at the same time present adhesions or bands of inflammatory tissues remaining after an attack of peritonitis, or in whom a congenital slit in the mesentery is found, such a case being cited by Henoeh (*"Vorlesungen über Kinderkrankheiten,"* Berlin, 1897). They are, in fact, examples of *internal strangulation*. A case of acute obstruction from flexion due to a drawing back and twisting of the intestine by an adherent Meckel's diverticulum is reported by Van Lennep (*Hahnemannian Monthly*, Oct., 1890). Laparotomy was performed on the sixth day of the obstruction and the child recovered. *Incarcerated and strangulated herniæ* are rare in childhood, but have been observed. *Foreign bodies* or masses of ascarides becoming firmly lodged in the bowel are also the cause of the obstruction at times.

Intussusception and the other forms of obstruction are surgical diseases, but as the medical practitioner is the first to see them it is eminently important that he should be thoroughly acquainted with their clinical course and be able to promptly recognize them. The prognosis only too frequently depends upon an early diagnosis. If the true nature of a case of intussusception or fulminating appendicitis be overlooked until sloughing of the bowel in the one case and septic peritonitis in the other have set in every chance of recovery, even in the event of an operation, will have been lost.

#### INTUSSUSCEPTION.

Intussusception is most frequently seen in children during the first year, and has been found to occur oftener in boys than in girls. Although diarrhoea with constant straining has at times seemed to be the exciting cause, it has occurred just as well during torpid conditions of the bowel.

Intussusception consists of the invagination of one portion of the intestine into another, most frequently the lower end of the ilium, together with the cæcum, into the colon. The invaginated portion produces a sausage-shaped tumefaction in

the region of the cæcum or transverse colon, often advancing over into the left iliac region. It may at times be felt in the rectum, even protruding therefrom for a considerable length.

Neither of these signs, however, may be discernible, particularly the tumor, which cannot be felt after the abdomen becomes much distended.

The onset is usually sudden, the symptoms being colicky pains, with vomiting and straining at stool. The lower bowel soon becomes emptied of its fæcal contents, after which passages of blood and mucus make their appearance. The vomiting becomes stercoraceous unless the obstruction is relieved, and the patient dies in collapse.

Spontaneous reduction or sloughing of the invaginated portion of the gut, and successful union with restoration of the lumen of the canal may occur in exceptional instances. Such a case has been recently reported by Steinmeyer. (*"Münch. Med. Wochenschrift,"* vol. xliii, 1896.) Stricture, however, follows nature's cure in the majority of instances.

The *prognosis* is grave unless the intussusception can be reduced within a reasonable time of its occurrence. Gibson (*N. Y. Medical Record*, July 17, 1897) estimates the mortality as 53 per cent. from a collection of 249 cases. It is claimed by many that operation should not be undertaken after twenty-four to forty-eight hours.

**Diagnosis.**—Symptoms of obstruction, together with the presence of the sausage shaped tumor in the abdominal cavity and in the rectum, bloody stools and active movements of the intestinal coils above the seat of obstruction and projectile vomiting are positive evidences of intussusception.

#### APPENDICITIS.

Appendicitis is seldom seen as early as intussusception, only exceptionally occurring during infancy, and rarely before the fourth year. The causes are the same as in adults. *Appendicular colic* is more common in children, however, than in adults, owing to the more patulous state of the open-

ing of the appendix into the cæcum, permitting the entrance of faecal concretions (*stercoraceous appendicitis*). These cases usually recover, as the appendix is able to empty its contents back into the cæcum more rapidly than in the adult. Every case of severe colic in a child under ten years, therefore, should be examined for tenderness over the base of the appendix, which, if present, would indicate inflammatory reaction.

It has been observed that appendicitis in children is more frequently of the *fulminating variety* than in adults.

The *catarrhal variety* is characterized by its mild course and absence of complications.

The *perforative variety* may, from the beginning, be accompanied by localized plastic or suppurative peritonitis, or without any warning an apparently mild case may suddenly perforate and set up a general septic peritonitis.

The clinical features of appendicitis are very characteristic, and cannot be more tersely or more clearly described than the following paragraph from Van Lennep's monograph ("Appendicitis," *Trans. of the American Institute of Hom.*, 1897) indicates :

"There is the history of improper eating, or perhaps exposure to cold, associated with the menstrual period in the female; occasionally overexertion, particularly in the sedentary, or possibly a direct traumatism. Then, what have been aptly termed the cardinal symptoms: (1) *Pain*, at first peri-umbilical or diffuse, but soon referred to the right iliac fossa, unless the appendix points elsewhere. (2) *Tenderness*, almost always present at the junction of the organ with the cæcum (McBurney's point); sometimes associated with other sore spots corresponding with distal lesions or their products. (3) *Muscular rigidity*, to corroborate tenderness, which may vary from a local or general board-like stiffness to an indistinct, circumscribed muscular tension, or a barely appreciable difference between the two recti at their costal margin. Besides this three-legged stool, as Hering would have termed



these cardinal symptoms, are the well-known initial concomitants: Sudden onset, the coated, flabby and indented tongue; the vomiting, which, when present, is from an overloaded or rebellious stomach; constipation, sometimes preceded by an irritative diarrhoea; distention, usually local in the early 'tympanitic tumor,' due to atony of the cæcum from an irritated appendix; and, lastly, as might be expected, a moderate temperature rise and pulse acceleration."

**Diagnosis.**—With the presence of the above symptoms the diagnosis of appendicitis is not difficult. From intussusception it is differentiated by the absence of projectile or stercoraceous vomiting, bloody stools and intestinal tumor. Furthermore, in intussusception there are active movements of the intestines, while in appendicitis "actively-moving intestinal coils are not seen or felt, and gurgling is scanty or absent" (VAN LENNEP). Referred pain in the right iliac fossa is not uncommon in pneumonia of the right base in childhood. It is caused by irritation of the trunks of the last dorsal spinal nerves at the site of their exit from the spinal foramina. When there have been recurring attacks of appendicitis we can often palpate the thickened appendix through the abdominal wall.

**Treatment.**—Although every case of intussusception and appendicitis is by no means a surgical one, and careful prescribing together with the proper management of the case yield the best results in non-perforative appendicitis and in reducible cases of intussusception, still the physician must be constantly on the alert and learn to recognize the indications calling for operation.

In appendicitis, Van Lennep says: "My working-plan regarding operation is about as follows: In a severe attack, characterized by sudden onset, and particularly by intense cardinal symptoms, with or without corresponding concomitants, operation should be undertaken at once. In a milder seizure, the more common form, recovery may be looked for, with the hope of an interval operation. In deciding the ques-

tion of persistence in such cases I have come to rely more than ever on the twenty-four hour limit, and I believe that whenever doubt or would-be conservatism has induced me to delay, I have had cause to regret the inaction. Benign cases will show signs of improvement within twenty-four hours, while unfavorable cases, requiring operation, usually grow worse during this time, and become dangerous from the possibility of perforation with septic infection of the peritoneal cavity."

In the case of intussusception, reduction should be attempted as soon as possible by means of taxis carefully applied and inflation of the bowels with fluids or air. This is unsafe after the third day, by which time firm adhesions will have formed. The child is best anæsthetized, a soft-rubber catheter introduced into the rectum, and by means of a fountain syringe, held at a height of three feet above the child's buttocks, from a pint to a quart of warm normal saline solution may be allowed to run in gradually, while the patient is inverted and the abdomen manipulated to aid in the reduction. Henoeh recommends the use of ice-water. The operation is not without danger, rupture of the bowel having occurred where the height of the fluid was four and one-half feet (*Harrington, Boston Med. and Surg. Jour.*); here, however, the injection was used after the third day.

Failing in this, laparotomy is the last resort, and the earlier performed, the greater the chances for recovery.

For the relief of the troublesome vomiting, lavage of the stomach is highly recommended by many writers.

Early in the attack such remedies as *Bell*, *Nux vom.*, *Colocynthis*, *Cupr.* and *Gelsem.* may be of decided benefit. On the whole, *Bryonia* seems best indicated to check the plastic inflammation.

In appendicitis an opening of the bowels must be obtained as soon as possible by the judicious use of enemata, to be followed by a saline purge when improvement begins. A liquid diet is also imperative. For relief of pain and control of the

inflammation there is nothing so efficient as the ice-bag. Heat is contra-indicated, acting as a poultice and favoring necrotic changes in the appendix.

*Nux vomica*.—This is the most important remedy in the early stage, indicated by coated tongue, nausea and vomiting, colicky pains in abdomen, constipation with urging to stool, abdomen tender and bloated.

*Belladonna*.—Intense pain and sensitiveness in the right ileo-cæcal region, cannot bear the weight of the bedclothes or to be touched. There is high fever, flushed face, vomiting, patient lying motionless on back with right leg drawn up.

*Bryonia*.—Inflammatory stage. *Bryonia* covers the pathological condition more closely than any other remedy, and its cardinal symptom, pain aggravated by motion together with inflammatory fever, thirst and constipation, are almost invariably present.

*Dioscorea*.—Severe pain in abdomen, beginning in region of umbilicus and extending to right iliac fossa, which becomes sensitive to pressure. The pain is constant, twisting in character, and becomes worse in paroxysms; constipation and thirst.

*Mercurius sol.*—Painful tumefaction in right ileo-cæcal region; tongue broad and flabby, showing imprints of teeth; constipation; fever, worse during night, with sweat, which gives no relief.

*Rhus tox.*—Hard, painful swelling in right side of abdomen; patient lies on back with legs drawn up; great restlessness, but cannot lie on either side; tongue dry and red, with triangular tip; typhoid state.

*Sulphur* is a most useful remedy during the intervals to prevent recurrence and remove the products of inflammation at the end of an attack.

When suppuration is suspected *Hepar* will be indicated. Other remedies which have proven useful are *Arsenicum*, *Carbo veg.*, *Lycop.*, *Plumbum*, *Veratr. alb.* When these remedies are indicated septic peritonitis, as a rule, is present, making the prognosis most grave.

## INTESTINAL PARASITES.

The parasites infesting the intestinal tract of children, which are of practical importance from the clinical standpoint in this country, are two round worms, the *oxyuris vermicularis* and *ascaris lumbricoides*, and two tape-worms, *tænia saginata* and *tænia solium*.

Regarding the disturbances produced by the presence of these parasites nothing definite can be said, as it cannot be determined positively that a child has worms until they are discovered in the stools, or a microscopical examination of the fæces reveals the eggs of whichever species may be present. Fortunately they are rare, and the symptoms attributed to worms are in many cases dependent upon some other disturbance of the general health, commonly chronic intestinal catarrh or rickets, and at other times even more serious organic disease, which is often overlooked from the convenience of classing all children's complaints under the heading of "teething" and "worms."

One who comes in contact with a large number of children soon learns to recognize the lack of dependence one can place in symptoms popularly considered diagnostic of worms. Time and time again an anthelmintic brings forth no worm where these symptoms exist, and, on the other hand, children in apparently good health, without a single suspicious symptom, will pass worms in great numbers.

In the case of the *oxyuris vermicularis*, pruritus ani at bedtime, recurring regularly each night, in some cases even violent pains in the rectum; enuresis, and in male children erections with consequent masturbation, are frequent symptoms. These worms also migrate into the vagina in females, inducing leucorrhœa and masturbation. They are found in the fæces, and can be detected at night emerging from the anus.

The *ascaris lumbricoides* is to be suspected when there are attacks of colicky pains; intestinal catarrh with loose stools

or mucus in the fæces; nausea and vomiting not due to disordered stomach; irregular appetite; pale countenance with dark circles under the eyes; dilated pupils; itching of the nose; gritting of the teeth; restless sleep with starting, and atypical febrile disturbances. All of these symptoms may, however, be traceable to other conditions; and here, again, the worm or its eggs must be discovered and other diseases excluded before a positive opinion can be given. We meet with children who have repeatedly passed ascarides and yet present none of the above symptoms, while others have many of the symptoms but no worms. It is claimed that an extract of ascarides injected into rabbits proves fatal, and, therefore, that the parasites secrete toxic substances, to which are due the nervous symptoms of helminthiasis (DEMATTEIS).

Dr. La Fuente (*Presse Medical*) considers attacks of intestinal colic coming on suddenly, seizing the child usually at play, and confined to one part of the abdomen, and bilateral narrowing of the field of vision as pathognomonic signs of ascarides. During the attacks of colic the abdomen is quite sensitive to palpation at the seat of the pain. This symptom I have been able to verify. We must, however, be on our guard and not mistake an attack of appendicitis for this condition. The narrowing of the field of vision is at times so pronounced, according to the above observer, as to be readily detected by passing the finger to and fro before the child's eyes. While these symptoms should be considered as presenting strong evidence of helminthiasis, still we cannot give a positive opinion until every effort has been made to discover the worms or their eggs in the stools, as other conditions may produce similar disturbances.

*Tape-worms* are the least common variety in children, but cases are occasionally seen. They are usually unsuspected until segments of the worm are passed, although tape-worms may produce marked anæmia in the young. In every case of pronounced anæmia the stools should be examined for parasites.

**Morphology.**—The *oxyuris vermicularis*, also known as the thread-worm and seat-worm, is a small, whitish, thread-like worm, attaining a length of 10 m.m. in the case of the female, the male being 4 m.m. The female has an acuminate tail, while the male is blunt (Fig. 31). They infest the lower ileum and upper colon, often in great numbers. The females prefer the cæcum and the colon, according to Zenker and Heller (Ziegler, "*Text-Book of Pathological Anatomy*"), and, when mature and egg-bearing, migrate into the colon and rectum to deposit their eggs, whence they also creep out of the anus at night. The eggs are oval, flat on one side and rounded on the other, and exceedingly small. Before they can develop they must first enter the stomach of some host, and it is quite likely that a child often reinfects itself by swallowing the eggs from its own colony of parasites.

*Ascaris lumbricoides*.—This is the common round worm, being cylindrical in shape, with tapering extremities and light reddish-brown in color. The female may attain a length of fifteen inches; the male eight to ten inches. The eggs are larger than those of the oxyuris, and possess a double shell, the contents being dark and granular. They measure about 1-340 inch in length (Fig. 32). The mature female sheds enormous numbers of these ova—according to Eschricht and Leuckart, 160,000 daily. The life-history of the ascarides is not fully understood. They infest mainly the small intestines, although they may be found at any point in the alimentary tract, sometimes even being vomited, and in rare instances inducing death by creeping into the ductus communis chole-  
dochus or into the larynx.

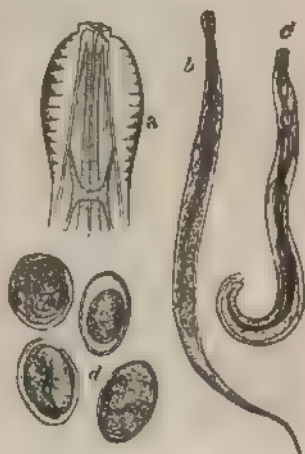


FIG. 31. OXYURIS VERMICULARIS. (JAKSCH.) a, HEAD; b, MALES. c, FEMALES. d, EGGS. (SIMON, *Clinical Diagnosis*.)

*Tænia saginata*.—This form of tape-worm is derived from beef, and is perhaps the most commonly met with variety in this country. It has a square head, with four suckers, but no hooks (Fig. 33). It may attain a great length, and the segments are very numerous, and longer than broad. The life-history of the worm is as follows: After the eggs are discharged into the intestinal tract by the mature segments, they reach the alimentary tract of oxen grazing on pastures where these infected stools have been passed. Here the embryos are

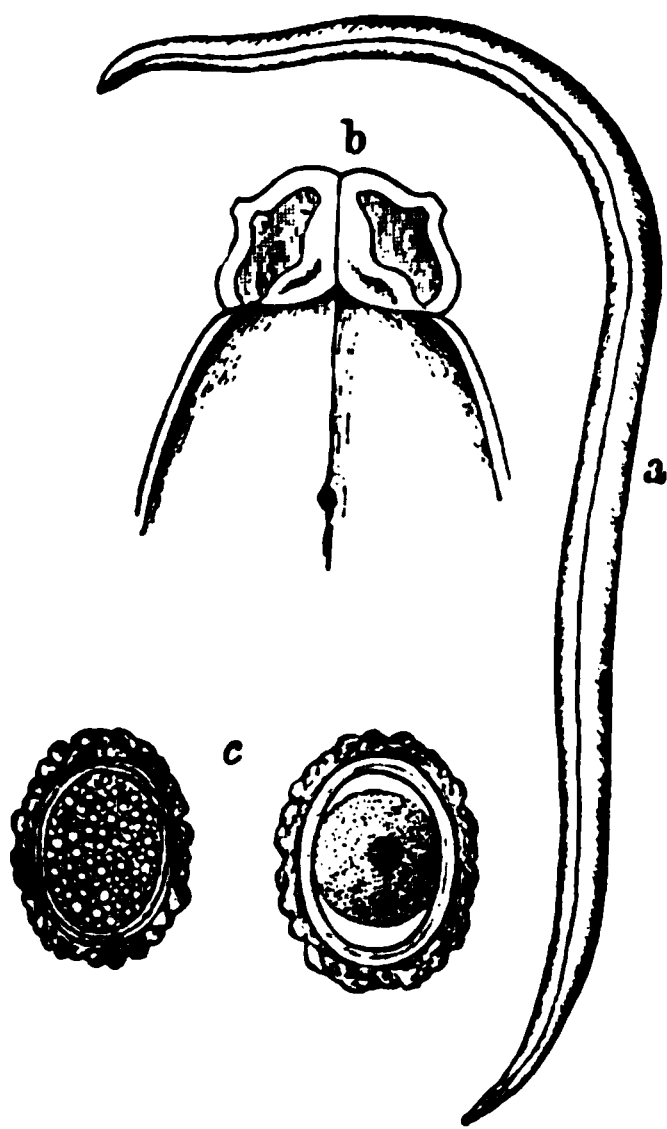


FIG. 32.—*ASCARIS LUMBRICOIDES*  
(V. JAKSCH.) *a*, WORM, HALF  
NATURAL SIZE; *b*, HEAD,  
MAGNIFIED; *c*, EGGS.  
(SIMON.)

liberated and find their way into the muscular tissue throughout the body, and sometimes into various organs, where they become converted into the cysticercus, or larval form. If this cysticercus is eaten with raw or insufficiently-cooked meat, the capsule is destroyed by the digestive juices and the contained scolex liberated, which attaches itself to the mucous membrane of the small intestine, where it soon develops into the fully-matured form by segmentation.

*Tænia solium*, also known as the armed tape-worm, is derived from pork, and differs from *t. saginata* in being equipped with a set of hooks besides two pairs of suckers (Fig. 34).

This parasite is also much smaller than the other variety and is less frequently encountered in the United States.

**Treatment.**—In the case of oxyurides nothing appears to give more desirable results than the daily use of warm salt-water enemata. They must be given as high as possible, for these worms are not confined to the rectum alone, as has been



pointed out. Owing to the possibility of reinfection, scrupulous cleanliness of the child must be observed. *Lard* may be applied to the anus at night to relieve the itching, or, what

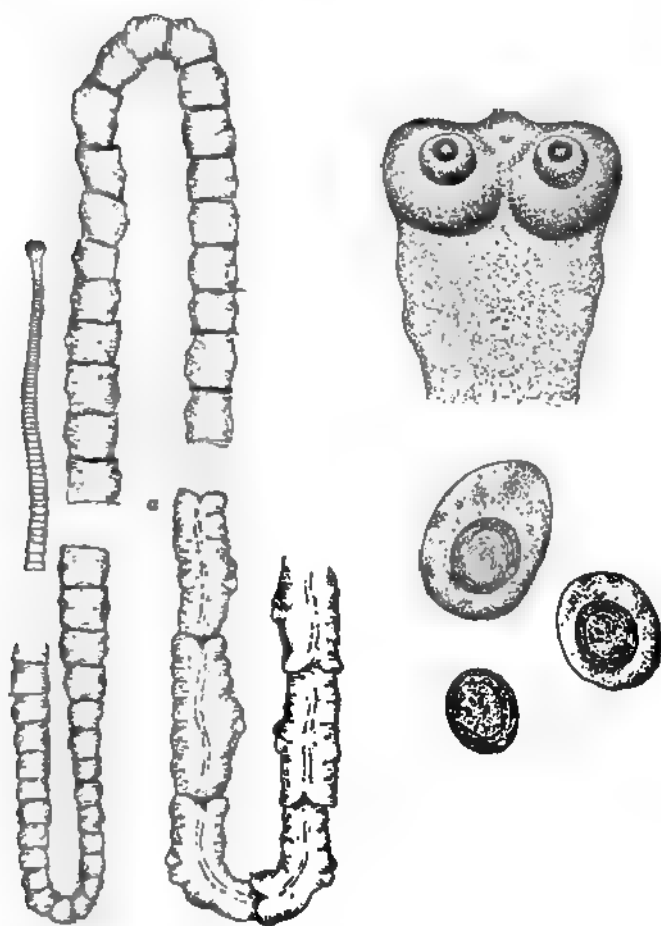


FIG. 33.—*TÆNIA SAGINATA*. THE SEGMENTS ARE NATURAL SIZE; HEAD AND EGG MAGNIFIED. (SIMON.)

acts better, *Carbolated vaselin*. For internal administration *Teucrium* is recommended, although *Cina* or *Sulphur* may be called for in some cases. "The nightly restlessness and

intolerable itching which they produce as long as they exist are almost always relieved by *Acon.*"—(C. G. R.) *Naphthaline* may be used in suppositories (see below).

*Ascarides* are usually promptly expelled by the administration of *Santonin*. This is best given in the first decimal trituration, a two- to five-grain powder, according to the age of the child, three times daily, followed, on the morning of the third day, by a laxative, if necessary.

*Naphthaline* is recommended as an anthelmintic, its most decided power being over the oxyuris, although it is also capable of removing round worms. One to five grains, according to the age, is administered on an empty stomach, followed by a cathartic, such as castor oil. In the case of seat



FIG. 34.—HEAD OF TÆNIA SOLIUM, AFTER LEUKHART. (SIMON.)

worms a rectal suppository of *Naphthaline* (two to five grains) may be tried after a salt enema. This is preferable to its internal administration.

One of the following remedies will be useful to remove the intestinal catarrh which often remains for some time after the worms have been expelled.

*Cina.*—Sickly, anæmic children, with pale face, dark rings under the eyes; irritable, cross disposition; great nervous irritability, waking from sleep in terror; variable appetite; picking at the nose; constipation and itching at anus; feverish during night; milky urine.

*Spigelia.*—Colicky pains in region of navel, usually worse evenings and accompanied by palpitation of the heart, twitch-

ing of the facial muscles or strabismus ; anæmia, with greenish hue of the skin ; intestinal catarrh.

*Stannum*.—Chronic indigestion, moaning during sleep, sluggish disposition ; “it prefers to lie on its stomach, to relieve the abdominal suffering” (FISCHER). *Stannum* is said to kill the ascarides when used in the lower triturations, after which they can be expelled by a purgative.

*Sulphur*.—Pale, sickly look ; eyes sunken, with blue margins ; canine hunger ; empty, faint feeling before dinner ; alternation of constipation and diarrhœa ; itching about anus ; emaciated, dirty-looking children, who are filthy in their habits.

For the expulsion of the tape-worm the *oleoresin of felix mas* is the most certain agent. A capsule containing fifteen minims is given on an empty stomach in the morning, and an hour later one to two teaspoonfuls of *laxol* may be administered. Should this not be successful, a larger dose must be used, preceded by a saline purgative, which has a tendency to remove all mucus from the intestinal tract, and thus fully expose the head of the worm to the action of the vermifuge.

## CHAPTER X.

### DISEASES OF THE PERITONÆUM.

#### ACUTE PERITONITIS.

Acute peritonitis is rare during childhood; it is most frequently seen in the new-born as a result of infection through the umbilical cord, or after the fourth year, when appendicitis begins to play a prominent rôle in the diseases of the abdomen. The other cases of peritonitis may be classed as primary and secondary, of which the latter is by far the more common. The primary variety develops suddenly from exposure to cold or traumatism, and in some instances a primary *pneumococcus* peritonitis is found, as a rule, coexisting with meningitis, pleurisy, pericarditis or lobar pneumonia (polyserositis of infants), although it is quite possible that the peritoneal infection is secondary to the above conditions in most instances, especially in the case of pneumonia and pleurisy.

Secondary peritonitis is more common and more easily explained, as it results from the extension of an inflammation from other parts or through infection. Under the latter heading comes peritonitis attending perforative appendicitis, perforating empyema, perforation of the intestine during typhoid fever, suppurating mesenteric glands, pyelo-nephritis, etc.

**Pathology.**—Pathologically, three forms of peritonitis exist, namely, fibrinous, or plastic; sero-fibrinous, and purulent. In the first stage there is injection of the peritonæum, with loss of lustre of its surface, due to the deposit of a fibrinous exudate and infiltration of the membrane, together with destruction of the endothelium. This is followed by serous exudation, varying much in quantity in different cases. The fluid is turbid from the presence of leucocytes, and contains flakes of fibrin; it may also contain red corpuscles. When the exudation is scanty, adhesion between opposing serous

surfaces takes place; when profuse, it accumulates in the peritoneal cavity, seeking its most dependent portions, thus lessening the liability to adhesions. In septic cases pus is formed from the beginning, while a sero-fibrinous inflammation may become purulent through an increased migration of leucocytes. Pneumococcus peritonitis is purulent throughout its entire course.

**Symptoms.**—The onset of peritonitis is usually abrupt, and, as it is often a complication of other diseases, we must be prepared to recognize its early symptoms. Sudden development of sharp, cutting abdominal pains, rise of temperature to 104° F. or over, rapid, thready pulse, distended, hypersensitive abdomen, vomiting, and obstruction of the bowels or diarrhœa are the classical symptoms pointing to this affection.

With the progress of the disease the abdomen becomes distended and tympanitic, the abdominal walls rigid, and the patient lies on his back with the legs drawn up, to relieve the tension. Respiration is thoracic and is rapid and shallow, the pulse is rapid and thready, the features are pinched, the lips and extremities cyanotic, and pain is usually severe. Although diarrhœa may be present, there is most commonly a constipated condition of the bowels, and in septic cases obstruction due to intestinal paralysis, indicated by vomiting; uniform distention of the abdomen, with cessation of all gurgling sounds, is the usual condition. These cases rapidly go on to fatal collapse, although early operative interference will occasionally save a life. In the very young all forms of peritonitis are fatal; in older children the non-purulent form is not so unfavorable.

**Treatment.**—To relieve the pain, *Bryonia* and hot fomentations should be prescribed, and, although rest is a great essential in the treatment of peritonitis, it is better to keep the bowels open than to paralyze them by the use of *Opium*. Adhesions are less likely to occur, and fatal obstruction is not invited if we avoid such treatment.

If the vomiting becomes troublesome all food must be with-

held, and nothing but cracked ice or a little hot tea allowed; champagne is very useful in such cases. Lavage also stands highly recommended for this complication.

The remedies most applicable to the early stages are *Acon.*, *Bell.*, *Bry.*, and *Ferrum phos.* After these will follow *Apis*, *Canth.*, *Mercurius corr.* and *Rhus tox.* when exudation sets in, *Sulphur* occupying a prominent position as an agent to absorb the exudate in the later stages. In grave cases, or in the advent of complications, we may find it necessary to employ one of the following remedies: *Arsenicum*, *Camph.*, *Carbo veg.*, *Lachesis*, *Lycop.*, *Nux vom.*, *Opium* and *Verat. alb.*

*Acon.*—After exposure to cold; hot, dry skin; rapid, hard pulse, with high fever, great restlessness and anxiety. Cutting and darting pains in bowels or burning in the abdomen.

*Apis.*—Exudation; burning, stinging pains; scanty urine; loud, piercing shrieks and cerebral symptoms; pneumococcus peritonitis coexisting with meningitis.

*Arnica.*—After traumatism, in early stages.

*Arsenicum.*—Later stages, impending collapse. Great anguish; clammy perspiration; the patient feels cold, and complains of burning pains in abdomen; restless tossing, thirst, obstinate vomiting, distention of the abdomen and cold extremities.

*Camphor.*—Collapse.

*Cantharis.*—Intense inflammation, pinched features, rapid, feeble pulse, cutting and burning pains.—(GOODNO.)

*Carbo veg.*—Great distention of the abdomen, with paralysis of the bowels. Extremities cold up the knees; collapse.

*Lachesis.*—Great hyperæsthesia of the abdomen; complicating gangrenous inflammation of the appendix; loquacious, adynamic fever.

*Merc.*—When the exudate tends to become purulent, indicated by chilliness, followed by sweat; starting in sleep; cachetic expression; foul breath; emaciation; obstinate vomiting.

*Nux vom.*—Threatened paralysis of the bowels; belching;

constant urging to stool without relief ; vesical irritation ; obstinate constipation and vomiting ; chilliness from uncovering ; abdomen painfully distended.

*Opium*.—Paralysis of bowels or antiperistaltic action ; incessant vomiting, distention of abdomen, somnolence and stupefaction ; warm sweat.

*Rhus tox.*—The *Rhus tox.* patient prefers to lie on his back with the legs drawn up, although the pains make him very restless. There is delirium at night, great prostration, and a brown tongue with red tip.

*Sulphur*.—To hasten resorption of exudate.

*Veratr. alb.*—Cold sweat on forehead, vomiting and purging, small rapid pulse ; great thirst, but drinking aggravates ; anxiety, prostration and high fever.

#### CHRONIC PERITONITIS ; TUBERCULOUS PERITONITIS

Chronic peritonitis is, in the majority of cases, of tuberculous nature, and when tuberculosis must be excluded the condition can be traced to a traumatism (Hench), or to an inflammatory condition or neoplasm of the abdominal viscera, excepting in the case of foetal peritonitis, which is syphilitic (Silbermann). These infants are mostly born dead, although they may survive with permanent peritoneal damage. Tuberculous peritonitis is usually secondary to intestinal tuberculosis, infection taking place from caseating mesenteric glands ; primarily it may develop as the acute miliary type. It frequently develops after weaning, especially when milk from a tuberculous cow has been fed to the child.

**Symptoms.**—The disease presents itself under several different types, more or less characteristic.

The *acute miliary tuberculous type* presents the symptoms of acute peritonitis and is rapidly fatal, although remissions may occur.

The *ascitic type* may be idiopathic or tuberculous. It is accompanied by an exudate of serum, or a purulent fluid in case of admixture of septic organisms. Idiopathic ascites



has been observed in girls at the time of puberty ; the condition disappeared after the appearance of the menses (QUINCKE).

In the *adhesive type* there is a matting together of the intestines by dense bands of plastic exudate. Caseous masses are found in the mesentery and in the bands of fibrous tissue, and a firm mass is produced which is difficult to entangle after the abdomen has been opened. It eventually produces obstruction of the bowels. Felt through the abdomen, the mass gives one the impression of a neoplasm. The *fibro-plastic variety* presents adhesions together with fibrinous exudation, which may undergo caseous degeneration and induce ulceration into neighboring organs. Fæcal fistulæ forming in the neighborhood of the umbilicus are not uncommon in these cases.

The *course* is slow and may terminate in spontaneous recovery, especially in the fibro-plastic type, although death may result from extensive adhesions of the abdominal viscera. The ascitic form frequently recovers under proper treatment, but a general infection is always to be dreaded.

**Diagnosis.**—The presence of fluid in the abdominal cavity, a well-defined nodular tumefaction due to adhesions and organized exudate, or a sense of roughness imparted to the examining hand when palpating and rubbing the surface of the peritonæum against the viscera, are physical signs to be relied upon. The nature of the peritonitis must be decided by the age, diathesis, presence or absence of tuberculosis in other organs and the history of the case. The abdomen is uniformly distended and fluctuating and shifting dulness in the flanks can be elicited in ascites without adhesions. Ridges and irregular contour of the abdomen suggest adhesions while stationary dulness speaks for sacculated fluid. In the adhesive variety an irregular, tumor can usually be palpated. Chronic obstruction of the bowels gradually develops when the adhesions constrict the gut.

In the presence of marked ascites, hepatic disease must be

excluded before a diagnosis of peritonitis can be made with certainty. In peritonitis the fluid contains more albumin and is of a higher specific gravity than in cirrhosis of the liver or other circulatory obstruction; besides an abundance of cellular elements will be found in inflammatory serum. A predominance of mononuclear elements speaks in favor of tuberculosis. We may also be able to find the tubercle bacillus.

**Treatment.**—Owing to the favorable results obtained by laparotomy, every doubtful case should receive the benefit of an exploratory incision, which may materially help the patient, if it will not result in a cure. The ascitic variety is the one especially benefited by laparotomy and evacuation of the fluid; in the others it is of doubtful value. Sutherland (*Archives of Pediatrics*, Feb., 1903) is not in favor of surgical intervention as in forty-one cases observed by him the results of medical treatment of all varieties was much better (70 per cent. recovered) than of surgical treatment (50 per cent. recovered, 50 per cent. died). In connection with this the patient must be carefully fed, receiving highly-nutritious diet and cod-liver oil, besides remedies of known value in tuberculosis and acites. As constitutional remedies, *Arsenicum jod.*, the *Calcarea*, *Sulphur*, *Silicea* and *Iodoform* are to be selected from.

## CHAPTER XI.

### DISEASES OF THE RESPIRATORY TRACT.

#### SPASM OF THE GLOTTIS.

Spasm of the glottis, or *laryngismus stridulus*, is a neurosis which manifests itself as a contraction of the muscles narrowing the glottis, with resulting embarrassment of breathing. This affection has received many names, and has been confused with other affections from which it is entirely distinct; thus, the *asthma of Millar* must, according to his description, be classed as spasmodic croup, and the *thymic asthma of Kopff*, if indeed such an affection can be established clinically, must also remain a distinct malady.

The principal *cause* is rickets, craniotabes being especially associated with the development of these symptoms.

Age and sex also play an important rôle in the etiology; the greatest number has been observed in children under one year of age, but the first dentition period may be considered as including most cases. As regards sex, males furnish fully two times as many cases as females.

Peripheral reflex irritation or fright usually acts as an exciting cause. The *attack* begins suddenly, often during the night, but not necessarily so. The child starts with great embarrassment of breathing, attempting to inspire, which is only accomplished by interrupted efforts, and is accompanied by a wheezing or squeaking sound. For a moment the child presents the picture of one being suffocated, when suddenly the spasm gives way with a forcible, crowing, inspiratory effort, and an attack of crying ensues. The *prognosis* is favorable under ordinary circumstances, but when associated with severe cases of rickets it has frequently proven fatal.

**Congenital stridor** of infants is a condition which may be mentioned in connection with glottic spasm. Its true nature

is not fully understood. Robertson considers it a paralysis of the crico-arytenoideus posticus, while Thompson and Turner look upon it as imperfectly coördinated spasmodic action of the muscles of respiration, *i. e.*, choreiform respiratory spasm. It occurs in young infants, and may exist from birth. The breathing is harsh and noisy. This noisy breathing may persist during sleep. The rhoncus is inspiratory and is loudest over the larynx.

Constitutional treatment must be instituted between the attacks, paying especial attention to rickets when this is present.

For the attacks there are several remedies highly recommended as specifics, but nevertheless it is necessary to differentiate individual cases.

*Sambucus*.—This remedy was first recommended by Hahnemann. According to Searle, "Burning, red, hot face, hot body, with cold hands and feet *during sleep*. On awaking the face breaks out into a profuse perspiration, which extends over the body and continues, more or less, during the waking hours; then, on going to sleep again, the dry heat returns (NICHOL, "*The Larynx and Trachea in Childhood*").

Another remedy of definite clinical value is *Chlorine*, administered in the first centesimal dilution, prepared freshly from a saturated solution of the gas in water. This remedy was introduced by Dunham, who made the following distinction between it and *Sambucus*—under *Chlorine* there is difficulty in expiration, none in inspiration; *Sambucus* has the reverse (*Ibid*).

*Belladonna*.—This is the principal remedy when its well-known cerebral and circulatory symptoms are present.

*Gelsemium* is highly recommended by Hale as a palliative.

Beside these may be mentioned, as other leading remedies, *Aconite*, *Arsenicum alb.*, *Cuprum*, *Grindelia robusta*, *Ignatia*, *Iodium*, *Sanguinaria*. Of the last named Nichol writes: "My own experience leads me to look upon *Sanguinaria* as being the Imperial Guard of all the remedies for spasm of the glottis. . . . I give it in the form of an acetous syrup."

## ACUTE CATARRHAL LARYNGITIS; SPASMODIC CROUP.

This form of croup, which must be distinguished from *true croup* or *pseudo-membranous laryngitis*, is a common affection of childhood, being a catarrhal inflammation of the lining membrane of the larynx associated with spasmodic action of the interior laryngeal muscles, thus giving the disease a paroxysmal character.

The anatomical and physiological peculiarities of the larynx and nervous system of young children, namely, the relative smallness of the larynx and rima glottidis, the great vascularity of its lining mucous membrane and the heightened reflex excitability of its nerve-supply, offer a ready explanation for the frequency of this malady during childhood and the peculiar type it assumes.

**Etiology.**—The chief exciting causes are exposure to cold, draughts or wet weather; acute indigestion and direct irritation, such as the inhalation of irritating vapors. Attacks occur more frequently during the winter and early spring than in the milder and dryer season. Beside all this, however, there seems to be an individual predisposition to the disease, often hereditary, or simply a catarrhal tendency or nervous temperament. Male children are more frequently attacked than females, and the age at which it is most likely to occur is between the second and third year.

**Symptomatology.**—The characteristic feature of spasmodic croup is its paroxysmal nature. The child may have been apparently well during the day, but towards night a ringing, metallic cough—sometimes before retiring, at other times not until the child has been put to bed—makes its appearance. There may be, instead, a slight hoarseness; but notwithstanding this, the child usually falls asleep, and sleeps soundly, only to suddenly awaken at midnight, or shortly before, with all the characteristic symptoms. The breathing is much oppressed, inspiration almost impossible, much prolonged, and accompanied by a harsh, rasping sound, while with expira-

tion there is a ringing, metallic (croupy) cough. The child is much alarmed, exceedingly restless, and the face presents a picture of anxiety and distress. Cyanosis and recession of the supra-clavicular and supra-sternal spaces is marked, if the attack be long-continued and severe ; often the sufferer grasps at the throat in the effort to relieve the obstruction.

The attack may last from a few minutes to an hour or longer, not, however, in one continued degree of severity. A second milder attack is likely to occur during the same night, and on the following two or three nights it may again be looked for with all probability. A moderate amount of fever, together with some catarrhal secretion, is usually present. The condition rarely results fatally.

A more severe form of *acute laryngitis* often confronts us and makes it difficult to decide whether the case be one of catarrhal or croupous inflammation. In these cases there is fever ; continuous hoarseness ; more or less dyspnoea and paroxysms of suffocative cough, together with abundant tenacious mucous secretion. So eminent an authority as Holt tells us that it is at times impossible to differentiate such a condition from true croup and that at the autopsy one is surprised at not finding membrane when such a laryngitis has proven fatal.

**Diagnosis.**—*Pseudo-membranous croup* is the most important condition, and often the most difficult one from which false croup is to be differentiated. In the absence of grave constitutional symptoms ; *complete relief* of all symptoms between attacks, which are always *paroxysmal* and most likely to occur shortly before midnight ; absence of exudation in the pharynx or upon the tonsil, and the presence of *hoarseness rather than suppression of the voice*, eliminate true croup with a fair amount of certainty. Again, auscultation of the larynx, revealing a dry, wheezing or hissing respiration and a hoarse-sounding, croupal cough, is, according to Trousseau, not a sign of exudation in the larynx, but rather one of its absence. In severe cases of *acute catarrhal laryngitis* the

fever is higher than in diphtheritic croup and there is no exudation to be seen in the pharynx. A laryngoscopic examination reveals the epiglottis, the arytenoids and the vocal cords deeply congested and swollen and covered with mucous secretion instead of membrane.

*Laryngismus stridulus* is to be differentiated from mild attacks, which can usually be done readily by a careful comparison of the two ailments.

**Pseudo-Membranous Laryngitis**, being almost invariably a laryngeal diphtheria, will, for the sake of convenience and clinical importance, be described under the subject of "Diphtheria."

**Treatment.**—During an attack the child should be supplied with steam inhalations as soon as possible, which may be accomplished by simply holding a basin of hot water before it, or, in case a croup-kettle or a steam spray are accessible, a tent should be constructed over the child and the stream directed under the same. If this does not offer sufficient relief a cold pack should be applied to the throat. Care should be exercised not to allow the child to inhale the vapor too hot for fear of aggravating the symptoms.

Remedies are both useful to mitigate the severity of the paroxysm and to overcome the tendency to recurrence.

*Aconite*, *Hepar* and *Spongia*, as recommended by Boënnighausen, are of exceptional service when given successively, although it will be found usually that one begins in its pathogenesis where the other leaves off, and it is therefore wisest to continue the single use of each of these remedies as long as it seems indicated.

*Acon.*—High fever, dry skin, great restlessness, nervous temperaments; after exposure to cold winds or draughts; checked perspiration.

*Acetic acid* is a valuable remedy in croup, especially when there is an accumulation of mucus in the larynx. A few drops of the acid added to the water feeding the steam spray makes a useful adjuvant. The indications under which it



does most good are the following: "Croup, especially when the face is bright red. (Diluted in water, ten drops in a tumbler of water with some sugar, a teaspoonful every hour or two.)" (*Hering's Condensed Mat. Med.*)

*Bell.*—Barking cough; pre-paroxysmal symptoms of attacks; child wakes suddenly; great vascular excitement; *rawness* and *pain* in larynx, with hoarseness.

*Spongia.*—"Its most remarkable therapeutic virtue is to cure croup. Among other symptoms, it is indicated in this disease by difficulty in breathing, as though a plug had lodged in the throat, and as though the larynx were so constricted that breath cannot pass through it."—(HAHNEMANN.) "The sawing respiration of this remedy is also characteristic. The aggravation is in the evening; *Hepar* in the morning."—(HERING.)

*Hepar.*—Deep, rough, barking cough; rattling of mucus in larynx and trachea; laryngeal symptoms remaining after the paroxysm.

*Potassium Permanganate of Potash*, in the experience of Dr. B. H. Sleght (*Hom. Eye, Ear and Nose Journal*, June, 1901), is a specific for the croup paroxysm given in teaspoonful doses of a cherry-red aqueous solution.

*Phosphorus.*—This remedy is recommended highly by several observers, often acting curatively when the above remedies have failed to give decided relief. It is especially useful for the hoarseness and bronchitis remaining after the attack.

*Ipecac* and *Lobelia* are extensively used by many physicians in this affection, and undoubtedly yield excellent results, provided they happen to suit the case.

#### ACUTE BRONCHITIS.

Acute catarrhal bronchitis is one of the common ailments of childhood, seen especially in the rachitic or those in whom malnutrition and anæmia are a prominent feature. Children who are closely confined, either in the poorer crowded quarters or in nurseries insufficiently aired and sunned, are particu-

larly susceptible to bronchitis, for which reason most cases prevail during the winter months and early spring.

Secondarily, bronchitis accompanies measles, whooping-cough, influenza, typhoid fever and several others of the infectious fevers almost unfailingly; its pathology and symptomatology are the same in these cases as in the primary variety.

Several varieties of acute bronchitis are to be recognized. The *mildest form* is simply an acute catarrhal condition, afebrile in its course and unaccompanied by constitutional disturbances. Baginsky prefers to call it *bronchial catarrh* in contradistinction to actual bronchitis. It is very prevalent among infants during the colder months of the year, and seems to be dependent upon atmospheric changes and constitutional predisposition.

*Acute febrile bronchitis* is infectious in origin, is accompanied by constitutional symptoms, and tends to spread to the finer ramifications of the bronchial tree, setting up suffocative symptoms—*capillary bronchitis*. When the process invades the pulmonary parenchyma, which takes place both by continuity of structure and by the formation of independent foci of solidification through the agency of micro-organism, we are confronted by a *broncho-pneumonia*.

*Pseudo-membranous bronchitis*, or *fibrinous bronchitis*, is in the majority of cases due to the diphtheria bacillus and may follow this disease. I have seen it in conjunction with pneumonia. The pus organisms may also induce fibrinous exudation upon the bronchial mucosa. A chronic form of obscure origin is to be encountered.

**Pathology.**—As in the case of spasmodic croup, a catarrhal inflammation of the bronchial tubes during infancy is of more serious import and accompanied by more suffocative symptoms than a similar condition in adult life. The greater vascularity and looseness of the mucous membrane, and the relatively smaller size of the air-vesicles and smaller amount of breathing-surface in the infantile respiratory tract, are the reasons

for these attacks assuming so dangerous a course. Outside of its tendency to spread to and involve the finer ramifications of the bronchial tree, acute catarrhal bronchitis presents nothing apart from the same process in adults. In fatal cases the mucous membrane appears swollen, injected, ecchymosed, and covered with mucus and purulent secretion. In the larger tubes the lining membrane alone is affected, while the smaller and finest ones are involved throughout their entire thickness in the inflammatory process. The lungs are usually emphysematous, from dilatation of the air-vesicles and choking up of the capillary tubes with secretion. Areas of atelectasis are also encountered.

Every grade, from simple hyperæmia of the mucosa with desquamation of epithelial cells up to the highest type of inflammatory reaction with infiltration of the sub-mucous tissue; necrosis of the epithelium and croupous exudation upon the surface of the membrane will present itself according to the severity and nature of the infection. The mucous membrane is covered with a tenacious secretion rich in pus corpuscles. Dilatation of the bronchi is a common result of severe bronchitis in children.

**Symptomatology.** --Bronchitis may run a mild or a dangerous course. In the first instance there will be a slight fever, cough, which at first is dry and irritating in character, later becoming loose and accompanied by rattling of mucus in the larger tubes. Some soreness in the region of the bifurcation of the trachea may be present, but the child evinces no great degree of pain or discomfort, and within a week or less the attack is over.

When the smaller tubes, however, become involved, the case presents an entirely different aspect. There is marked dyspnœa, imperfect aeration of the blood and enfeebled circulation, higher fever ( $103^{\circ}$  to  $104^{\circ}$  F. or over), and the chest abounds in subcrepitant and sibilant râles, besides coarse râles of mucus in the larger tubes. The child is exhausted from incessant cough and carbonic acid poisoning, and the

cough is too feeble to expel the mucopurulent secretion blocking the air-vesicles and bronchioles. It becomes dull and apathetic, even comatose, the pulse rapid and thready or imperceptible, and death, sometimes preceded by convulsions, terminates the scene.

This severe type, described as **capillary bronchitis**, is, strictly speaking, a broncho-pneumonic process, and it is impossible to draw a sharp line of distinction between an acute spreading bronchitis and a pneumonia. As stated above, the pulmonary parenchyma soon shares in the inflammatory process both through continuity and contiguity of structure and therefore these cases present bronchitis, peribronchitis and lobular inflammation.

**Diagnosis.**—In bronchitis the percussion-note never becomes altered unless emphysema, atelectasis or other complications develop during its course. In mild cases there are at first dry râles, followed by large moist râles, with here and there a sibilant and small moist râle, all best heard posteriorly. In the second variety *subcrepitant* and sibilant râles, general in distribution, with large moist and dry râles in the large tubes and trachea, and areas of dullness, with diminished respiratory murmur, indicating collapse of air-cells, may be elicited. Hyper-resonance, resulting from vicarious emphysema, is difficult to identify in children, as the normal percussion-note is in itself highly resonant.

Sufficient dilatation of some of the bronchi (*bronchiectasis*) to produce physical signs may result. In such cases bronchial breathing may be heard over the dilated bronchus and a tympanitic note can be elicited by percussion. The sputum is purulent and separates into a purulent sediment superimposed by a fluid and frothy layer.

**Treatment.**—In mild cases of bronchitis it is often advisable to keep the child out in the fresh air as much as possible, instead of rigid confinement to the bed or nursery. The predisposition to bronchitis must be overcome by cold sponging, plenty of out-of-door exposure, and the correction of the

underlying diathetic condition with appropriate remedies and diet fat being especially beneficial.

Severe cases of bronchitis should receive all the care and attention accorded a case of pneumonia.

Remedies are numerous, the most useful, however, judging from the frequency of their successful employment, being *Acon.*, *Bell.*, *Bry.*, *Ipecac.*, *Merc.*, *Puls.*, *Rhus tox.*, *Tartar emet.* and *Sulphur*.

Beside these the *Calcareas*, *Cham.*, *Ferrum phos.*, *Hepar*, *Hyos.*, *Lycop.* and *Phos.* are often indicated in individual cases.

In the early stages *Acon.*, *Bell.*, *Bry.*, *Cham.*, *Ferrum phos.* and *Mercurius* must be differentiated.

*Aconite* has high fever, dry skin, no chilly feelings as in *Mercurius*, nor disposition to moisture of the skin, as in *Belladonna*, which has a dry, distressing, paroxysmal cough, usually worse towards evening. *Belladonna* is looked upon by some as a specific. The old school resort to its use largely. Its usefulness cannot be disputed, but I see no reason for pushing the drug to its full physiological action.

The greatest usefulness for *Bryonia* seems to be to loosen the cough when the same shows no disposition to become so, remaining deep and hollow, apparently coming from the epigastric region, aggravated by motion and often accompanied by pain. *Scilla* is also strongly indicated by painful cough; it is, however, a more severe type than *Bryonia*, there being cyanosis and failing circulation, owing to extension of the process into the finer tubes.

*Cham.* suits mild cases of tracheo-bronchitis in the early stages; the cough is excited by attempting to use the voice, and the child is fretful and cross.

*Ferrum phos.*—Often preferable to *Aconite* in cases characterized by marked dyspnoea right from the beginning, with rapid progress, soon assuming the capillary variety. The cough is short and dry, often paroxysmal, and when expectoration appears it is streaked with bright blood. Well suited to rachitic subjects.

*Mercurus*.—" *Mercurius* corresponds with the whole course of a severe attack of bronchitis, even better than *Belladonna*. There is a violent fever, the temperature is very high, there is a great disposition to sweat without obtaining any relief from it ; in contradistinction to *Belladonna* there is a constant alternation of chills and heat, with a remarkable sensitiveness to the most trifling changes of temperature (BÆHR, *Science of Therapeutics*). " Tongue thickly coated yellow ; diarrhœa ; cough dry, worse evening until midnight ; dyspnœa ; expectoration tenacious.

*Lobelia inflata*.—"Think of lobelia in asthenic bronchitis of children with profuse secretions, and difficulty in removing the accumulations; also if there is a sense of oppression and feeling of dulness." (" *Medical Advance*," July, 1898, T. G. ROBERTS.)

As the cough becomes loose, *Ipecac*, *Pulsatilla* and *Tartar emet.* or one of the *Calcareas* will be required. For the therapy of the severe types and complications the reader is referred to the article upon *Broncho-pneumonia*.

In a case of *pseudo-membranous bronchitis* I obtained excellent results from *Phos*.

#### CHRONIC BRONCHITIS.

Chronic bronchitis may result from repeated attacks of acute bronchitis, or, more commonly, follow upon an attack of whooping-cough, measles, or other acute illness, in which there is offered predisposition to the development of bronchitis. In infants, rickets or simple malnutrition lay the foundation for chronic bronchitis, while in older children the scrofulous diathesis is found. As a secondary disease, it accompanies tuberculosis, organic heart disease and Bright's disease.

The important *pathological conditions* are thickening of the mucous membrane, with areas of superficial ulceration, weakening and irregular dilatation of the bronchial tubes, and more or less extensive vesicular emphysema.



The important *symptoms* are cough and expectoration, the characteristic condition being, that notwithstanding the long continuance of these symptoms, the general health rarely suffers to a marked degree. Naturally, these children are not up to the normal standard of health, as the etiology of the affection indicates; at the same time there is no pronounced wasting or suffering induced by the disease.

The cough is loose, usually paroxysmal, and may become dry and teasing at times. It is generally worse in the morning, and the expectoration of large quantities of offensive muco-pus on rising, associated with localized gurgling râles, is strongly indicative of *bronchiectasis*.

The *course* is a slow one at the best, and cases may be apparently cured in the summer only to have a relapse during the winter. Nevertheless, the *prognosis* is good, the condition being much more amenable to treatment than in adults for the reasons that the tissues are more regenerative and the disease less frequently dependent upon an incurable associated condition.

**Treatment.**—An equable, moderately warm and dry climate is desirable; the mountainous pine regions are especially beneficial. Tonic treatment must be instituted in all cases—baths, fresh air, exercise and a highly-nutritious diet being the essentials.

Of the greatest importance in these cases it is to search for and correct any abnormality in the nose and throat. Septal deflections, spurs and polypi are frequent sources of irritation but more commonly adenoids and enlarged tonsils will be found. Enlargement of the lingual tonsil is often responsible for persistent winter coughs and should be looked for.

*Hepar sulph.* I have found of especial benefit for the paroxysmal cough coming on at night. A powder of the second or third decimal trituration will usually relieve these attacks with astonishing promptness.

*Pulsatilla*, of course, is indispensable for the loose cough with profuse easy expectoration of yellowish or yellowish-



green muco-pus, having a tendency to become tighter and more troublesome at night. This remedy acts very satisfactorily with *Hepar*, and I frequently employ it during the day, giving a dose of *Hepar* at night.

*Lycopodium* is particularly useful for the recurrent type of bronchitis, in which the patient is seldom free from a troublesome cough, "catching cold" on the slightest provocation. "Cough dry, day and night, in feeble, emaciated boys."—(C. WESSELHOEFT.) Lithæmic subjects; acid dyspepsia; cough ending with a loud belch.

*Sulphur*.—Rarely will a case be found in which *Sulphur* is not at one time or another indicated. Especially in the scrofulous or rheumatic type of constitution will it be found useful. It has not proven of much use where emphysema was present; but where there is a large amount of tenacious mucus, mixed with lumps of pus, of foul taste and odor, it seems particularly applicable. There may also be attacks of oppression of breathing, in which the patient gasps for air.

*Tart. emetic*.—Useful in recent cases, with loud râles in the larger tubes, and dyspnœa with the cough.

The *Calcareas* are especially called for upon their diathetic indications.

*Calc. carb.*, beside its characteristic sweat, large belly and glandular enlargements, will be indicated by loose cough, with expectoration of yellowish, sweetish mucus, or dry, teasing cough, with dyspnœa and palpitation of the heart from slightest exertion. *Calc. phos.* is more suited to the purely rachitic with diarrhœa, or cases of simple malnutrition.

*Silicea*.—Emaciated children, tuberculous diathesis; night-sweats; profuse purulent expectoration; skin dry and scurfy; hectic fever; bronchiectasis; lack of normal body-heat, with constant chilliness. The cough is aggravated from cold drinks, and is deep and distressing.

Beside these it may be necessary to resort to one of the following remedies for special conditions and symptoms:

*Ars.*—Emphysema ; dyspnœa.

*Carbo veg.*—Hoarseness ; chronic spasmodic cough remaining after whooping-cough. General loss of vascular tone of the entire mucous membrane of the respiratory tract.

*Iodium.*—Especially indicated in dark-complexioned, emaciated children. Ravenous appetite without a corresponding gain in weight ; enlarged bronchial glands. The *Iodides* are particularly useful in the bronchitis accompanying phthisis.

*Kali bichromicum.*—Tough, stringy expectoration ; cough excited by eating. Bronchitis after measles.

*Kali hydriod.*—Syphilitic cases.

*Stannum.*—Bronchial dilation, with excessive purulent expectoration ; weak feeling in chest. *Stibium iodid* is also an excellent remedy for bronchiectasis.

#### ASTHMA.

The majority of cases of asthma occurring during childhood are of the catarrhal type, the asthmatic paroxysm accompanying a bronchitis or broncho-pneumonia. The typical spasmodic type as seen in adults is rare, seldom occurring before the sixth year, although mild asthmatic phenomena such as *bronchial spasm*, occurring with dentition ; *asthma dyspepticum* (HENOCH), due to indigestion, and *hysterical asthma* (*pharyngeal spasm* and *hysterical tachypnœa*), are frequently met with prior to this time.

Idiopathic spasmodic asthma is most probably a vasomotor neurosis intimately associated with the lithæmic diathesis, although the bronchial-spasm theory has still many adherents. Local irritation induced by pathological conditions of the nose and pharynx plays an important rôle as a reflex exciting cause. Personally I cannot accept any other explanation for the suffocative symptoms than that of turgescence and swelling of the mucosa. Asthma is, so to speak, a “hay-fever” of the bronchia. During attacks of asthma it has been possible to see the mucosa of the trachea and study its

condition. Freeman has seen it so swollen in cases of influenza accompanied by great dyspnoea that the lumen of the trachea was almost occluded.

**Symptomatology.**—The attacks occur suddenly, usually at night, the chief symptom being dyspnoea, accompanied by a dry cough and characteristic respiration. The inspiration is difficult, accompanied by recession of the soft parts of the thorax, and expiration is prolonged. The respiratory mur-



FIG. 35. SPASMODIC ASTHMA, ILLUSTRATING THE ACTION OF THE ACCESSORY RESPIRATORY MUSCLES AND THE DISTENTION OF THE LUNGS.

mur is diminished, and the chest abounds in sibilant and sonorous râles; wheezing may be heard at quite a distance from the patient. Cyanosis becomes pronounced if the attack is a prolonged one. The attacks may last from a few minutes to an hour or more, and generally cease suddenly with a free secretion from the bronchial tubes; they recur at intervals of days or weeks.

The *catarrhal form* is only the engrafting of the asthmatic element upon a pre-existing bronchitis or broncho-pneumonia, in individuals thus predisposed. At times these children are subject to pseudo-croup, the asthma seemingly taking the place of the former. During its entire course they are always more or less "wheezy."

The **diagnosis** depends upon a recognition of the nervous element in the case—the spasmodic and paroxysmal nature of these attacks, together with the characteristic physical signs of the diseases, namely, dyspnœa, cyanosis, diminished respiratory murmur, loud sibilant and sonorous râles, vesiculo-tympanitic percussion-note. When bronchitis or broncho-pneumonia coexist, their signs must be discounted.

**Treatment.**—The same hygienic measures recommended for bronchitis are applicable to overcome the tendency to recurring attacks of asthma. All foci of local irritation in the naso-pharynx or elsewhere should receive prompt attention.

Of equal importance, and in some cases the *sine qua non* for a cure, is the strict, systematic supervision of the diet of the patient. Exercise, personal hygiene and thorough ventilation of the sleeping apartment must be insisted upon.

The remedies most useful to mitigate the attacks are *Acon.*, *Ars.*, *Ipecac* and *Nux vom.*; beside these there are several others which are often prominently indicated. The interval requires constitutional treatment.

The inhalation of a *spray* containing a few drops of the tincture of *Ipecac* acts as a palliative during attacks. In some cases it is necessary to burn *Stramonium* leaves in order to make the suffering endurable.

*Acon.* is recognized by its well-known mental condition, feverishness, etc.; neurotic cases.

*Apis.*—When the attacks seemingly follow the recession of an urticaria, or alternate with the same. The chest feels bruised, and the attack ends with the expectoration of a large amount of frothy mucus and serum. It is a valuable remedy for the asthma of children.

*Ars.*—Paroxysms between midnight and daybreak; must get out of bed; great anguish and prostration; broncho-pneumonia.

*Ars. jod.*—Between the attacks.—(BELLVILLE.)

*Ipecac.*—Wheezing; constant cough, with subcrepitant râles all over chest; no phlegm yields, although the chest seems full. Gagging and vomiting; the child stiffens during the choking attacks; cyanosis and coldness of extremities.

*Lobelia.*—In connection with disordered stomach; weakness in pit of stomach; attack preceded by prickling sensation in extremities; distressing tightness across upper portion of chest.

*Nux vom.*—Asthma dyspepticum; attacks in morning; irritability and constipation.

*Pulsatilla.*—Cough, becoming dry toward night, with dyspnœa; inability to lie down; chilliness; mild, tearful disposition.

*Tart. emet.*—Rattling of mucus in larger bronchial tubes, with wheezing, great dyspnœa, and threatened collapse. This is a most valuable remedy for the catarrhal form of asthma, when there is a large secretion of mucus, together with pronounced dyspnœa.

#### ACUTE BRONCHO-PNEUMONIA.

Broncho-pneumonia, also described as *catarrhal* and *lobular pneumonia*, is one of the most common diseases of childhood, presenting a mortality rate only exceeded by diarrhoeal diseases, and being particularly prevalent before the fourth year of life. As the name indicates, it is a pneumonic process associated with inflammation of the bronchial tubes, in reality an extension of the latter condition into the walls of the terminal bronchi and surrounding end alveoli.

**Etiology.**—A primary and a secondary broncho-pneumonia are to be distinguished. Primarily broncho-pneumonia occurs with especial predilection in those debilitated by previous ill-

nesses, or in the rachitic and syphilitic. Atmospheric changes are the chief exciting cause, as the greater prevalence of this disease during the winter months and early spring clearly indicates. Primary broncho-pneumonia is rarely seen after the fourth year, being practically a disease of early childhood.

Secondary broncho-pneumonia accompanies and complicates the acute infectious fevers, prominently the exanthemata, diphtheria, whooping-cough and influenza, a class of diseases in which bronchitis is a frequent accompaniment.

The latest bacteriological researches indicate that primary broncho-pneumonia is nearly always due to the *pneumococcus*, while secondary broncho-pneumonia results from a mixed infection, in which the *streptococci* of suppuration play the most important rôle. When complicating diphtheria the *Klebs-Löffler bacillus* is the excitant of the pathological process. Pearce (*Jour. Boston Soc. Med. Sciences*, June, 1897) found in sixty-two cases of this class the Klebs-Löffler bacillus fifty-two times, the streptococcus pyogenes twenty-seven times, the staphylococcus pyogenes aureus eleven times, staphylococcus pyogenes albus once, pneumococcus once. In seventeen cases the Klebs-Löffler bacillus occurred alone; in seven the streptococcus pyogenes. In the other cases there was almost always a combination of these varieties, with, however, a preponderance of the cocci. In summing up, he remarks that where a local or general infection existed the pneumonia was due to the same micro-organism, but where the condition was a chronic or non-infectious process it was generally due to the pneumococcus. The investigations of Prudden and Northrup (*Amer. Jour. Med. Sciences*, June, 1889), and those of Neuman (*Jahrbuch für Kinderheilk.*, vol. xxx., 1889), and others practically lead to the same conclusions. In pure pneumococcus cases the temperature is generally uniformly high, while in those due to the streptococcus wide fluctuations in the fever are more likely to occur.

The etiological relationship of influenza to broncho-pneu-

monia has been carefully studied by Prudden (*Influenza and its Complicating Pneumonia*, *New York Med. Record*, 1890) and Weichselbaum (*Wiener Klin. Wochenschr.*, 1890).

**Pathology.**—In the larger bronchi we encounter a superficial inflammation, while in the smaller tubes the entire wall shares in the pathological process, and we find here both bronchitis and peribronchitis. The characteristic lesions are in the air vesicles, which in typical cases are distended with cellular exudation. The cells are mainly swollen, desquamated epithelia with small nuclei. Red blood corpuscles and leucocytes are also found in variable number. Fibrin as a rule is scant; often entirely absent. The fibrin in these cases is difficult to demonstrate, as the threads are rendered indistinct through the presence of a large number of leucocytes. (ZEIGLER.)

In the alveolar septa and peribronchial connective tissue the blood vessels are distended with red blood corpuscles and these structures are infiltrated with large mononuclear leucocytes.

Taking into consideration the above histological changes in the pulmonary tissue it is clear why resolution is slow and why often it is delayed, leading to permanent tissue changes. On account of the coexisting bronchitis in the finer tubes, it is clear also why in the presence of much mucous secretion suffocative symptoms may arise (capillary bronchitis).

It may happen—and this is not uncommon during the second and third year of life—that a mixed form of pneumonia develops, in which one portion of the lung is the seat of typical catarrhal and interstitial inflammation while another portion is consolidated by purely croupous exudation without involvement of the alveolar walls and peribronchial tissue. These cases pursue more closely the clinical course of broncho-pneumonia than lobar pneumonia, but it requires microscopic examination to recognize the true character of the lesions.

Frequently small broncho-pneumonic areas representing consolidated alveoli may spread and become confluent, thus



invading an entire lobule and giving rise to a lobular pneumonic process. These lobular areas are in the majority of cases separated by streaks of uninvaded lung tissue, *i. e.*, lobules still pervious to air. An entire lobe may, however, become invaded, in which case we are confronted with a broncho-pneumonia of lobar distribution. (ZEIGLER.)

The exudate in some instances is hæmorrhagic in character. When resolution is delayed it frequently becomes purulent owing to the presence of a large number of leucocytes that have undergone degeneration.

Although the inflammatory process may become general, as is the case in bronchitis, still pneumonia tends to localize itself, in this way differing from the former condition. A localized lesion, therefore, is either pneumonia or tuberculosis, and rarely, if ever, bronchitis. Again, the temperature is higher and more persistent in pneumonia than in bronchitis.

As Holt points out, the term broncho-pneumonia is a generic one. It is therefore impossible to describe the entire group by a single case, even though such a case present the leading features common to this group of pulmonary inflammation. Broncho-pneumonia on the one hand may abort in the early stage before consolidation can be detected and thus run the course of a severe bronchitis, while on the other it may assume the characteristics of a lobar pneumonia. Again, instead of undergoing resolution the inflammatory process may continue and interstitial pneumonia be the result.

The pathological findings are by no means uniform and as Delafield has pointed out the consolidated lobules may bear no definite relationship to the bronchus leading to them. The inflammation is diffuse in character, and lobule after lobule may become consolidated without its communicating bronchus being simultaneously involved. The inflammation therefore travels through contiguity of structure as well as by continuity thereof.

In the early stage (*red pneumonia*) the lung is engorged and of an intense red color. On section, a bloody, frothy fluid exudes from the air cells. The bases are heavier and darker in color owing to hypostatic congestion. Consolidation has not yet taken place, but microscopic examination reveals cell-proliferation in the peri-bronchial connective tissue and septa and catarrhal and hæmorrhagic exudate, in the alveoli. The process may abort here, prove fatal, or go over into the stage of *mottled* or *red and gray pneumonia*, representing the fully developed process. By this time the consolidated areas may be felt as small nodules in the pulmonary parenchyma. Both the surface and the sections present a mottled appearance due to the admixture of consolidated (gray) and congested (red) areas. The process may involve an entire lobe or appear only in patches dispersed through the otherwise normal lung tissue. Wherever a bronchus has become occluded areas of atelectasis are seen. Such areas correspond always to a bronchus, but consolidated lobules, as has been pointed out above, do not.

If resolution be delayed or arrested, the so-called *gray pneumonia* is the result. In these cases the lung is somewhat enlarged, gray in color and extensively consolidated. Pleural thickening and adhesions are common. On section a mucopurulent exudate covers the cut surface. The bronchial walls and the interstitial tissue are hyperplastic and areas of atelectasis and compensatory and interstitial emphysema lie interspersed between the consolidated structure.

In the cases that recover the *termination* is resolution through expectoration and resorption of the exudate; in unfavorable cases suppuration; interstitial induration; gangrene.

Resolution may begin before consolidation can be detected. Ordinarily it is completed in from two to three weeks. When delayed, there is a strong tendency to incompleteness of the process. In recurring attacks, permanent interstitial changes are produced as a rule. Tuberculosis may be engrafted upon a pneumonia secondarily.

The *pleura* shares in the inflammatory process when the lesions are superficial. Fibrinous and fibro-purulent exudate is poured out upon the surface of the visceral pleura with the consequent development of adhesions and thickening. In some instances the pleuritic process is of equal moment with the pulmonary; these cases are described under a separate heading (see Pleuro-pneumonia).

**Symptomatology.**—Primary broncho-pneumonia begins as a bronchitis in the majority of cases; exceptionally the pulmonary changes develop at the same time or prior to, or even independently of, the former. Instead of advancing favorably as an uncomplicated bronchitis, there are added progressively increasing dyspnoea and rapid breathing, increase in fever and pulse-rate, and prostration.

Some cases begin abruptly with high fever, rapid breathing and pronounced nervous disturbances (toxæmia). They may prove fatal before any signs of pulmonary inflammation have had time to develop; even cough may be absent. At the autopsy the lungs are found intensely congested and more or less œdematous.

In young infants broncho-pneumonia may come on insidiously, fever being slight during the entire course. The main symptoms are cough and progressively increasing cyanosis and rapid respirations. As a rule, gastro-intestinal symptoms accompany the pneumonia. The prognosis is grave.

During the progress of the disease the child emaciates markedly and carbonization of the blood becomes apparent. The pulse is rapid and weak, and the heart may eventually fail in its work if the pulmonary obstruction be extensive.

Cough is a prominent symptom, at first being dry and later becoming loose, although this by no means indicates that the child is gaining relief, for the secretion may be beyond its control, acting as a mechanical obstruction to the air-cells.

The respiratory rhythm is changed in a characteristic manner, the recognition of which has always been to me a strong indication for pneumonia. Normally, inspiration and expira-

tion follow each other without interruption, after which comes a pause. In broncho-pneumonia inspiration is separated from the expiratory act by a well-marked pause, with no pause, however, between the expiration and inspiration. The reason for this change in rhythm is undoubtedly to bring the inspired air in contact with the pulmonary tissue as long as possible in order to overcome the carbonization of the blood; therefore the child rests rather before expiration than after it, no time being lost, thereby, to draw in a fresh supply of oxygen.

Respiration is often accompanied by fan-like movements of the alæ nasi and recession of the soft parts of the thorax, notably its lower portion, producing the peri-pneumonic groove of Harrison.

When broncho-pneumonia develops during the course of one of the infectious fevers as a complication, it is to be suspected from an increase in the fever; increased rapidity of breathing and pulse-rate; cough and dyspnoea, especially the latter.

Broncho-pneumonia tends to localize itself in certain areas of the lungs, in this way differing from simple bronchitis in which the process is general. General bronchitis, however, may accompany pneumonia. In the absence of definite signs of consolidation, the height and duration of the fever may be taken into consideration. The statement Cabot makes about broncho-pneumonia in the adult, namely, that the patient is too sick to have simply bronchitis, applies with equal force to children.

Broncho-pneumonia is progressive in its development, being slower both in its onset and in the formation and resolution of its pathologic products than lobar pneumonia. Its course sometimes extends over several weeks, and the tendency to chronicity is strong, especially in the scrofulous and tuberculous.

Meningeal symptoms are of common occurrence in the disease, sometimes being toxæmic in origin, at other times result-

ing from an active congestion of the meninges with serous effusion into the arachnoidal spaces. Here there is always hyperpyrexia and a very rapid course, and rachitic subjects seem most prone to develop this complication. Actual meningitis from infection of the brain with pneumococci is by no means rare. I have found it more frequently after broncho-pneumonia than after the lobar form.

Death results from respiratory or cardiac failure; sometimes from hyperpyrexia. Collapse is the commonest termination, although convulsions may appear to close the scene. The fulminating cases undoubtedly die from toxæmia.

The *prognosis* must always be guarded, as can be seen from the high mortality rate; it is especially grave when the child is very young and debilitated, or when the disease is secondary to a condition in itself dangerous. The pulse and respiration are the main indications of the child's condition, and although a high temperature is a bad omen, still it is not necessarily so unless it is continuous and proves itself beyond control. Rickets seems especially likely to invite hyperpyrexia. The soft condition of the chest-wall in rickets makes breathing very difficult in pneumonia and rachitic children stand the disease badly.

A grunting expiration is said to indicate atelectasis, but it is not necessarily a bad symptom, unless very pronounced and persistent. The cough is also a guide to prognosis; if it becomes weak and inefficient we must expect gradual suffocation, unless the exudation can be absorbed.

Children in whom the tuberculous diathesis is well marked are liable to the most serious consequences from an attack of broncho-pneumonia. An ordinary broncho-pneumonia will become tedious; the temperature remits, leading us to suspect a possible malarial condition, but the case continues, in spite of our best-directed efforts, toward a fatal termination. Primary tuberculous broncho-pneumonia is of slower onset and the temperature seems out of proportion to the physical signs that can be elicited. Besides, the tubercle

bacillus can be demonstrated in these cases. As infection generally spreads from the bronchial glands, the presence of subcrepitant râles localized in the region of the nipple on either side indicate the presence of lesions of this nature and offer strong presumptive evidence of tuberculosis. (HOLT.)

**Diagnosis.**—The physical signs are those of both bronchitis of the larger and smaller tubes, together with consolidation of scattered areas of pulmonary tissue of varying size and extent. They are best studied posteriorly, the child being held over the nurse's shoulder. Large and small moist râles; subcrepitant râles; tubular breathing and dullness over the consolidated areas large enough to convey these signs; diminished breathing over areas of atelectasis, and exaggerated breathing in the vicariously emphysematous lung are to be elicited. However, the irregular fever, dyspnœa, alteration in the respiratory rhythm and cough, and the detection of subcrepitant râles and areas of tubular breathing are usually quite sufficient, and often the only available signs upon which the diagnosis can be made.

From *croupous pneumonia* it is distinguished by its gradual onset, tedious course, bilateral distribution, and its occurrence in the very young and in the feeble, croupous pneumonia attacking those in apparently good health and of maturer age. The differentiation from *tuberculosis* has been given above. In *capillary bronchitis* there are fine moist râles generally distributed throughout the chest. There is, however, no sharp line of demarcation between the pathology of the two affections.

**Treatment.**—The child should be put to bed, and its position changed regularly to avoid adding hypostatic congestion to the already seriously crippled condition of the lungs. Infants can be taken up by the nurse during coughing paroxysms and held face downwards or on the side to facilitate the expulsion of the phlegm. The room must be faithfully ventilated, and a temperature of about 70° F., or slightly lower, is to be maintained. Beside this, it is essential to keep the



air moist in the immediate vicinity of the child, which is best accomplished by means of the croup-kettle or steam spray and a tent improvised over the bed.

Ordinarily, the fever is within the control of remedies and sponge-baths; indeed, an alcohol sponge-bath (one part alcohol to three parts of water) has a most decided effect upon the temperature, rapidly bringing it down to within a safe limit, at which point it is maintained for an hour or more. In fulminating cases, or such as do not respond to the above treatment, the graduated cold, full bath will be required. This is often a life-saver, not only reducing high temperature but also relieving pulmonary congestion and acting in a decidedly stimulating manner upon the heart and respiratory centres. When carbonization of the blood becomes manifest and the bronchial tubes become clogged with secretion, the alternate application of hot and cold packs to the chest should be resorted to. This is a most powerful respiratory stimulant besides acting as a derivative and relieving pulmonary engorgement.

*Oxygen inhalations* should never be neglected in serious cases. The mistake is to look upon oxygen merely as a *dernier ressort*; given in time, however, it is a powerful agent to save life. I give from one to two gallons (bagfuls) every hour, administered by holding a glass funnel attached to the tube from the water-bottle of the apparatus over the child's mouth and nose. I prefer this to a mask or inserting a glass tube into the nostril.

Plain woolen underwear, the weight conforming to the time of year, is all that is necessary to protect the chest, which, with the rest of the body, should be regularly sponged.

*Stimulants* can rarely be dispensed with, and they will be called for during certain periods in all bad cases. Above all, every effort must be made to keep up the nutrition of these little patients.

The remedies most frequently indicated in the early stages are *Acon.*, *Bell.*, *Bry.*, *Ferrum phos.*, *Ipecac* and *Scilla*; for



the later manifestations, especially the unfavorable symptoms likely to arise, *Tartar emetic*, *Phos.*, *Arsen.*, *Carbo veg.* and *Veratrum alb.* are called for.

*Aconite* should always be studied in comparison with *Veratrum viride* and *Ferrum phos.* All three are indicated early in the disease, when there is high fever and a teasing cough, with little or no expectoration—the stage of congestion. *Aconite* is distinguished by its great anxiety and restlessness, thirst, and aversion to being touched or moved, which induces suffering; *Veratrum viride* by its high arterial tension, bloodshot eyes and cerebral irritation; *Ferrum phos.* by the absence of either nervous erethism or high arterial tension and by its characteristic frothy, blood-streaked expectoration. It is particularly applicable to the rachitic diathesis.

*Arsenicum* is indicated by extreme prostration and restlessness; dyspnoea from the slightest exertion; thirst for small quantities of water, the mouth being dry and the tongue and lips cracked; diarrhoea; cold surface.

*Belladonna* is particularly valuable when nervous disturbances are pronounced. Its excellent effect in capillary bronchitis makes us think of it in pneumonia when the bronchial symptoms predominate. In oedema of the lungs *Atropia* is the sheet anchor. *Belladonna* is exquisitely homœopathic to the vascular engorgement and high temperature so prominent in many cases.

*Bryonia* is of the greatest service to loosen the cough, control pain, and check the extension of the process into the smaller tubes and promote the absorption of the exudation. It must be differentiated from *Scilla*, which is similar in many respects, but more suitable to grave cases marked by progressively-increasing prostration and dyspnoea; rapid, weak pulse; short, painful cough, causing the child to cry faintly after each paroxysm; in fact, it cannot be moved without giving it pain. In my experience, the younger the child, the more efficacious has been this remedy. Hale (*Practice of Medicine*) considers *Scilla* the remedy above all others

after *Aconite* and *Belladonna*, being in every respect homœopathic to broncho-pneumonia.

*Chelidonium* is recommended where the right side is chiefly affected, with associated hepatic disturbances. It has the faul-like motion of the *alæ nasi* so strongly indicative of *Lycopodium*. Personally I have no experience with it. Dr. Bigler considered *Chelidonium* very useful in capillary bronchitis. It was recommended by Teste as a specific.

*Gelsemium*.—Broncho-pneumonia complicating influenza; after sudden checking of perspiration; pain under *scapulæ*; drowsiness; soft, rapid pulse.

*Ipecac.* is the remedy where the bronchial element predominates and the chest seems literally filled with mucous secretion, subcrepitant râles being heard everywhere in abundance. The cough is troublesome and gagging, giving little relief. The secretion gradually collects to such an extent in the finer bronchi that suffocation becomes imminent. Here it differs from *Tartar emetic*, which represents a state of carbonic acid poisoning, in which mucus, collecting in the larger tubes, produces the characteristic rattling, or in which there is active pulmonary œdema.

*Lycopodium* is useful in broncho-pneumonia, its particular sphere being, so to speak, a "choked-up" condition of the entire respiratory tract. The nose is obstructed; the *alæ nasi* expand with each inspiration, which is often a purely sympathetic condition, not dependent upon marked dyspnoea. The cough is dry day and night, a few moist râles and some wheezing being heard over the sternum; swelling of the mucous membrane of the bronchi seems to predominate over secretion. Likewise the lungs may be much involved, without, however, much cough or secretion. The child is peevish and irritable, especially on awaking from sleep; the urine is scanty and deep red, and when passed often induces crying; all symptoms are worse in the afternoon and early evening.

*Phosphorus*.—Where consolidation predominates over the bronchial symptoms, together with active congestion, produc-

ing a tight, distressing cough; rapid, shallow respirations; tightness across the upper portion of the chest; blood-tinged expectoration; failing right heart. We are inclined to think of *Phosphorus* only in lobar pneumonia, but it is of equal value in the lobular variety when we have to deal with congestion, consolidation and toxæmia; in fact, the old school has for a long time prescribed *Phosphorus* as a nerve tonic in the adynamia of pneumonia.

*Sulphur* is similar to *Phosphorus* in respect to the consolidation, but it has a greater power of removing the same, *Phosphorus* mainly controls the vascular disturbance (unless pushed to produce fatty degeneration of the inflammatory products, which is not without danger). It is indicated in the later stages of broncho-pneumonia.

*Tuberculin* (KOCH) has been highly recommended for broncho-pneumonia. Dr. Mersch (*Jour. Belge d'Hom.*, 1894 and 1895) reports several cases in which relief was rapidly obtained from the sixth dilution. Dr. Arnulphy (*Clinique*, Feb., 1896) makes strong claims for the efficacy of *Tuberculin* in broncho-pneumonia, placing it above such remedies as *Ipecac.*, *Iodine*, *Tartar emetic* and *Phosphorus*. *Bacillinum* (BURNETT) is recommended by Cartier (*Trans. Internat. Hom. Congr.*, 1896) in respiratory affections characterized by *oppression* and *muco-purulent expectoration*; the dyspnœa results from pulmonary obstruction, caused by excessive secretion in the bronchi. In his opinion, these cases are non-tuberculous. He recommends the thirtieth potency, one dose every two to three days. *Aziare*, or *Avian tuberculin*, he has found useful in broncho-pneumonia following influenza and measles, accompanied by an incessant tickling cough, with closely localized pulmonary symptoms and emaciation—*suspicious bronchitis*—which causes apprehension of tuberculosis. Personally I have no experience with these products. It has seemed to me unnecessary to call upon such uncertain agents in the face of the all-sufficient array of well proven and verified remedies at our disposal. It is true, in tuberculosis a

serum may yet be prepared that will give positive results, but so far there is nothing absolutely certain with which I am acquainted.

#### CROUPOUS PNEUMONIA.

Croupous, or *lobar pneumonia*, is a primary acute infectious disease in which one or more of the pulmonary lobes are consolidated by a croupous exudation. Bronchitis may be associated, but it is not an essential condition as in broncho-pneumonia; besides the infection arises, so far as can be determined, primarily within the alveoli, being due to a specific micro-organism.

**Etiology.**—Croupous pneumonia is most frequently seen after the third year, and usually attacks those of previously good health, unlike broncho-pneumonia, which attacks with predilection those already debilitated or develops in conjunction with the acute infectious diseases. Exhaustion and exposure to cold act prominently as predisposing causes, for which reason genuine pneumonia is often seen to follow upon active play in cold weather when boys are likely to become overheated or become chilled from neglecting to dress properly. While the dry, cold months, particularly the early spring, furnish the largest number of victims, still pneumonia may be seen at any time of the year, like all other infectious diseases. Boys are more often attacked than girls, no doubt because they expose themselves more than the latter.

The sputum of pneumonia patients was long known to contain micro-organisms in abundance, but it was not until Fraenkel, in 1886, demonstrated the lance-shaped diplococcus named after him that the specific cause of the infection became established. Since then, however, it has been proven that other micro-organisms also may set up croupous inflammation in a pulmonary lobe or portion thereof. They are notably the pneumobacillus of Friedländer, the influenza bacillus, the typhoid bacillus and the staphylococcus and streptococcus. I have encountered the typhoid bacillus prac-

tically in pure culture in a case of pneumonia complicating typhoid fever. The influenza bacillus may associate itself with the pneumococcus and render the course of the disease more virulent and irregular. Jousset (*Revue Hom. Française*, June, 1901) has contributed most interesting observations to the literature of this subject.

The pneumococcus is found in great abundance in the alveolar exudate and may enter the general blood current, setting up a septico-pyemia or localized complications, notably pleural, meningeal and peritoneal inflammation.

**Pathology.**—In typical cases of croupous pneumonia one lobe is affected throughout its entirety. The most frequently consolidated lobe is the left lower; next in frequency come the right lower and the right upper lobes. The right middle and the left upper are least often attacked.

More or less plastic pleurisy is always associated, as is also bronchitis of the larger tubes. Membranous bronchitis seems at times to be due to the pneumococcus; I have seen it associated with pneumonia in one instance. When the left lower lobe is affected and the pleura is involved the process may spread to the pericardium. The pleural inflammation may become so prominent as to influence notably the clinical course of the disease.

At the onset of pneumonia, the *stage of engorgement*, the affected lobe is bright red, greatly congested and somewhat œdematous. The lung appears enlarged, as if inflated, and when the inflammatory exudate fills the alveoli and solidifies, the consolidated lobe is actually larger than normal, for which reason the area of dulness elicited by percussion may be of greater extent than the lobe normally occupies.

On microscopic examination the alveoli appear engorged, the bloodvessels encroaching upon the lumen of the same. A small amount of serum and leucocytes is now poured out, the exudation becoming more and more rich in cells and fibrin and more hæmorrhagic in character. It is at this time that the crepitant râle is most clearly heard. The alveoli eventu-

ally are distended to their utmost with red and white blood corpuscles and micrococci embedded in a stroma of fibrin. The fibrin also fills the lymphatics in the interstitial connective tissue, and it can be seen communicating by thin bands through the pores of the alveoli. This period represents the *stage of red hepatization*.

The color of the lung gradually passes from red over into gray, owing to the compression of the bloodvessels of the alveoli by the exudate and to the degeneration of the cellular elements. This represents the stage of *gray hepatization*. The exudate is now gradually removed by the lymphatics, some being expectorated after having undergone softening, and *resolution* is in progress. In normal cases resolution is complete and the lung is restored to its former condition.

During consolidation the lung is quite friable and cuts like liver. On the surface of the section small plugs of hardened fibrin filling the alveoli and independent therefrom are seen, giving it a granular appearance. In children this does not show as typically as in adults, owing to the lesser development of the air cells. At times, owing to a gradual spread of the process, all stages, that is, red and gray hepatization and beginning resolution, may be encountered in a cut of a single lobe.

When resolution is delayed it may terminate in suppuration with abscess formation, gangrene, caseation. Complete recovery is, however, the rule, excepting in cases complicated with pleural inflammation, in which it is quite common for an empyema to develop secondarily.

**Symptomatology.**—The onset of croupous pneumonia is rapid, and the course of the disease is characterized by its acuteness throughout; sudden onset, high temperature, with but slight remissions and terminating within from six to eight days by crisis, are the features of a typical case (Fig. 36).

The initial symptom is characteristically a chill, which may be replaced by a convulsion in young children; sometimes vomiting is the sole symptom. The temperature rises rapidly,

soon reaching a height of  $104^{\circ}$  or over; the pulse is rapid and full, and the respirations are notably increased, exceeding the normal ratio between pulse and respiration. Thus, with a pulse of 130 there will be 60 or more respirations, while the normal ratio is one respiration to four heartbeats. The temperature ranges between  $102.5^{\circ}$  and  $104^{\circ}$  F. or over. Remissions are more pronounced than in adults

Associated with the fever there is restlessness; dry, hot skin; headache and some delirium toward night, and a dry,

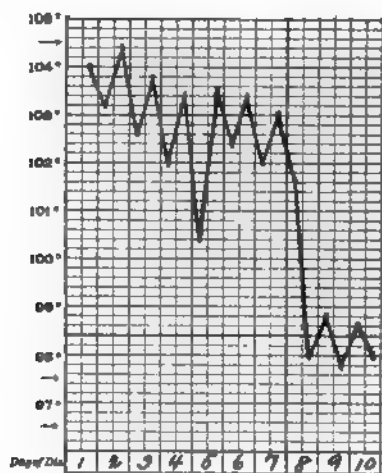


FIG. 36.—TEMPERATURE CURVE IN A TYPICAL CASE OF LOBAR-PNEUMONIA, SHOWING PSEUDO-CRISIS.

painful cough. Especially when there is considerable involvement of the pleura does this painful cough become conspicuous, it being very sharp and located at the seat of the inflammation. At times the pain is referred to the epigastrium, in which case it is due to irritation of the intercostal nerves, or it may indicate a complicating pleurisy or pericarditis. Pain in the right iliac region may also be complained of in pneumonia of the right base. When a child complains of



abdominal pain during the course of a febrile attack we should never neglect to thoroughly examine the chest.

Within from two to four days the process of consolidation is generally complete, as can be demonstrated by the dulness and bronchial breathing observed over the affected area. With the *crisis*, which may appear on any day from the fifth to the ninth, oftenest, however, on the seventh day, there is a marked amelioration of all symptoms. A profuse sweat accompanies this sudden fall in temperature, and at times, in-

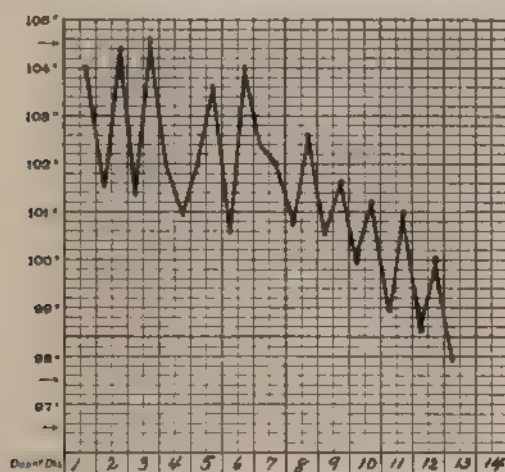


FIG. 37 —TEMPERATURE CHART OF A CASE OF REMITTING PNEUMONIA.

deed, there occur quite alarming symptoms of collapse, calling for immediate action. After the crisis the process of resolution becomes established, being completed in from five days to a week in the average case. I have seen every evidence of dulness and bronchial breathing disappear within three days after the crisis. A rise of temperature during this time—in other words, a post-critical rise—indicates the development of some complication, such as pleurisy, empyema, meningitis, pericarditis or the extension of the pneumonic process to

other portions of the lungs. A *pseudo-crisis* is common in children. It may occur as early as the second day, more commonly one or two days before the actual crisis. Termination by *lysis* is more common in children than in adults. Marked remissions in the temperature are also more common in children than in adults. When pronounced these cases are described as *remittent pneumonia* (Fig. 37).

The *blood changes* are important. While there is but a slight anæmia, leucocytosis develops to a marked degree. A pronounced leucocytosis indicates a severe infection in an organism capable of good reaction (DA COSTA). Leucocytosis offers a strong sign of differential diagnosis between pneumonia and such conditions as acute typhoid septicæmia, caseous pulmonary tuberculosis and serous pleurisy, it being absent in these conditions. It is of no value, however, in the differentiation of croupous pneumonia from broncho-pneumonia, empyema and meningitis (DA COSTA).

Many severe cases of pneumonia present so different a clinical picture from the group of symptoms above enumerated that they merit separate description, being classified into the following varieties:

**Cerebral pneumonia.**—This form is characterized by rapid onset with high fever, convulsions or vomiting, and a predominance of cerebral symptoms during the entire course of the disease. In other words, it is essentially a manifestation of pronounced toxæmia. In children over two years convulsions are not so common, these cases assuming more of a typhoid state, there being stupor, delirium, dry, brown tongue, involuntary stools. Symptoms simulating meningitis, such as sopor, strabismus, opisthotonos, slow, irregular pulse, retracted abdomen, dilated pupils, convulsions, are a frequent accompaniment of pneumonia, and there seems to be a close clinical relationship between pneumonia of the upper lobes and cerebral symptoms, notwithstanding that this is disputed by some competent observers. The pneumonic process is slow to develop in many cases, and often the consolidation

cannot be detected until four or five days after the onset, having begun centrally; for this reason it may be confounded with meningitis. The writer recalls a case of croupous pneumonia occurring in a child five years old which was diagnosed as meningitis by a most expert clinician until the detection of dullness and bronchial breathing in the right upper lobe on the fifth day, together with a disappearance of all serious symptoms by crisis on the seventh day, made it possible to recognize the true nature of the case. That these symptoms are toxic in nature there is little reason to doubt, but the possibility of a true purulent meningitis developing must never be lost sight of. This seldom, however, develops during the height of the pneumonia, a return of the fever with cerebral disturbance after the crisis being more likely to prove of serious import than the earlier manifestations.

Another form of pneumonia worthy of mention is the so-called **wandering pneumonia**, in which the pneumonic process spreads from its original seat to other portions of the lung, resolution going on at one point while a fresh invasion attacks another.

**Central pneumonia** is of especial interest from the diagnostic standpoint, as in these cases the process begins in the centre of a lobe, gradually spreading to the periphery. It is a patent fact that they cannot be recognized until there is sufficient consolidation to produce physical signs, and are frequently overlooked for this reason. Grave symptoms may exist with but a slight amount of consolidation, the toxæmia being entirely out of proportion to the existing lesion.

**Pneumonia with Gastro-intestinal Symptoms.**—Gaillard has shown that the enteric symptoms of pneumonia are due to the pneumococcus. Toxæmia, however, contributes its share in the production of pronounced gastro-intestinal derangements. I have on several occasions wrongly suspected intestinal auto-intoxication when the subsequent appearance of pulmonary signs and a crisis with amelioration of symptoms cleared up the case.

**Influenzal pneumonia** may be either due to the Pfeiffer bacillus or result from secondary infection with the pneumococcus. These cases begin as an influenzal bronchitis, during the course of which one or more pulmonary lobes become consolidated. The course is graver and more protracted than simple pneumonia. It is also liable to be followed by tuberculosis.

**Abortive pneumonia** is rare in children. Cases are encountered which terminate in from four to five days; they might be called *mild cases*. Again, the process may not go beyond the first stage, and although congestion of a single lobe and pneumococci in the sputum can be demonstrated, consolidation fails to take place, the process actually aborting, as other acute infections sometimes do. It is needless to say that the diagnosis is beset with great difficulty. There are also *fulminating* cases, terminating fatally in the first days.

**Typhoid-pneumonia.**—This misleading term refers to those forms of pneumonia in which the patient sinks into a typhoid state as the result of toxæmia. Instead of active brain symptoms being present as in cerebral pneumonia there is apathy and prostration; dry, coated tongue; tympanites with either obstinate constipation or diarrhœa; involuntary stools; muttering delirium; subsultus tendinum. Rose-spots, enlarged spleen and Widal reaction are negative. Typhoid fever, however, may begin as a pneumonia; in these cases a diagnosis can only be made when the last mentioned signs put in an appearance. In doubtful cases blood cultures should be made.

**Pleuro-pneumonia** is a form sufficiently distinct to merit separate discussion.

**Complications.**—A certain degree of *pleurisy* belongs to pneumonia. *Pleural effusions*, both serous and purulent, are, more strictly speaking, sequelæ; they are much more common in children than in adults. *Otitis* is common; it always produces an increase in and prolongation of the fever; not

necessarily pain. *Meningitis* is more likely to occur after the critical period; cerebral symptoms at the height of pneumonia are usually toxic and do not persist. *Pericarditis* is a grave complication; I have several times encountered it at the autopsy. It is seldom recognized *in vitam*. Other complications that may develop are endocarditis, peritonitis, gastro-enteritis, arthritis, septico-pyæmia.

**Physical Signs.**—The physical signs in lobar pneumonia vary with the different stages of the pathological process. The duration, clinical course and complications also modify these signs as well as the age of the child (anatomical peculiarities).

In the *first stage* inspection reveals a flushed countenance; rapid, shallow respiration and more or less pronounced dyspnoea. When the pleura is much involved the child turns over on the affected side. Dyspnoea may even progress to cyanosis; retraction of the supra-clavicular and supra-sternal regions; recession of Harrison's groove; fan-like motion of the *alæ nasi*.

On palpation the skin will be found hot and dry. The pulse is full and rapid. Vocal fremitus is not increased but coarse bronchial râles may be detected.

Percussion reveals dull tympany. This can be nicely demonstrated in children by gentle percussion as the chest-wall is still resilient.

We are dependent upon auscultation for a pathognomonic sign. This is the *subcrepitant râle*—a fine, crackling sound, produced at the end of inspiration by the separation of the walls of the air cells which at this stage contain a sticky exudate. As these râles may remain confined to a limited area and disappear after several hours, they are readily overlooked.

Friction râles, pleural in origin, are frequently heard. An interesting observation has been made by Shaw (*Archives of Pediatrics*, Aug., 1903), who found that the crepitant râle and friction sounds can be distinctly heard over the abdomen when the lower lobes are affected.

*Second Stage.*—With the completion of consolidation vocal fremitus is increased over the affected lobe and percussion dullness becomes pronounced. The area of dullness apparently covers a larger area than the anatomical boundaries of the lobe allow for. This is explained by the fact that the croupous process distends and enlarges the lobe. When pleural effusion takes place the lower portion of the dull area be-



FIG. 38. LOBAR-PNEUMONIA IN A CHILD FOUR YEARS OLD. THE DULL AREA IS OUTLINED AND CORRESPONDS TO THE LEFT LOWER LOBE. AT (X) FRICTION SOUNDS ARE HEARD AT (O) BRONCHIAL BREATHING AND BRONCOPHONY.

comes flat. It is not uncommon to hear friction sounds in the lower part of the chest, posteriorly and laterally, in pneumonia in this region. The adjoining normal lung, through compensatory emphysema, may give the vesiculo-tympanitic note.

Auscultation reveals bronchial breathing and bronchophony.



The respiratory murmur in the unaffected lung is harsh and loud, often greatly interfering with a proper study of the consolidated area. It is undoubtedly more difficult to outline an area giving bronchial breathing in the child than in the adult.

*Third Stage.*—As resolution sets in and the exudation begins to soften, crepitation reappears (*crepitatio redux*). Moist râles are usually added and considerable of the exudate is coughed up. Bronchial breathing persists longer than actual consolidation; so also dulness. This is no doubt due to the congested state of the pulmonary tissue. For this reason it is possible to demonstrate abnormal physical signs for a week or longer after the crisis. We must, however, regard with suspicion the persistence of pronounced dulness and diminished or absent respiratory murmur after pneumonia. Such a condition on closer investigation will be found to indicate most likely a sacculated empyema.

The physical signs of *pleuro-pneumonia* are described under that affection.

**Prognosis.**—In infants the prognosis is unfavorable. Robust children from three to ten years old recover as a rule. In fact, the mortality rate at this period of life is surprisingly low. The season of the year and the nature of the epidemic affect the prognosis. The association of influenza is unfavorable.

Of primary importance in gauging the prognosis is the degree of toxæmia. This seems more important than the extent of the pulmonary involvement or the height of the fever. Naturally, the spread of the disease to adjacent portions of the lung is unfavorable. The heart holds out better than in the adult because the child's circulatory apparatus can adapt itself to increased circulatory obstruction better than the adult's. The association of bronchitis, however, adds materially to the danger of the attack. Pronounced cerebral symptoms are also grave.

The majority of deaths occur at the height of the disease.



When death occurs later it is the result of one of the above mentioned complications.

**Diagnosis.**—Whenever we are confronted with an acute condition of sudden onset with high fever preceded either by a chill, vomiting or a convulsion, we should first examine the throat. Finding nothing specific here it behooves us to examine the chest most thoroughly.

It may be that at this early stage we may discover subcrepitant râles in one of the bases or in the right upper lobe and possibly a friction sound. The following day, together with a continuance of the high temperature and in older children the complaint of intense headache and pain in the side or epigastrium, we will find the evidences of beginning pulmonary consolidation. Even should we not be able to demonstrate the physical signs, as in central pneumonia (rare), still, the sudden disappearance of all symptoms at the end of a week or less justifies us in diagnosing pneumonia.

The conditions from which genuine pneumonia is to be differentiated are *broncho-pneumonia*, *pleurisy*, *meningitis* and *caseous tuberculosis*. I will not again go over the symptoms deciding the diagnosis. Suffice it to say, *broncho-pneumonia* is essentially bronchial in origin, both etiologically and pathologically, and that its course is long and protracted, independent of complications. In *pleurisy* the physical signs are essentially different and the onset gradual. The fever is not so high and terminates by lysis. Besides, primary pleurisy with effusion is rare in children, but pleuritic inflammation and the exudation secondary to pneumonia is common.

In *meningitis* symptoms are continuous and protracted. Death is practically always the termination excepting in the epidemic cerebro-spinal variety. Meningitis complicating pneumonia occurs in the later stages of the disease; cerebral symptoms occurring at the height of pneumonia are toxic and disappear by crisis or even before the crisis. Besides, they never attain the character of a true progressing meningitis.

*Typhoid fever* beginning abruptly may cause confusion. The absence of leucocytosis and the later appearance of rose spots, the Widal reaction and enlarged spleen positively identifies it.

*Acute caseous pulmonary tuberculosis* may set in with a chill and uniformly consolidate an entire pulmonary lobe within a remarkably short time. The temperature will run high and the entire clinical picture be identical with that of croupous pneumonia. Crisis does not occur, however, and eventually softening and break down of pulmonary tissue sets in. Elastic fibres and tubercle bacilli are to be detected in the sputum at this time, confirming the diagnosis. The most experienced are deceived, however, in the early stage of such a case.

#### PLEURO-PNEUMONIA.

In a certain number of cases of pneumonia (6.8 per cent. in Holt's series of 398) pleurisy exists at the same time with the pneumonic process and to such an extent as to give the condition distinct clinical features. The pleural inflammation is chiefly plastic in nature and the amount of serum poured out is relatively slight; never to the extent seen in a primary pleurisy. At the autopsy we will find the pleural surfaces matted together and covered with a thick, yellow, plastic exudate that can be readily scraped off and from the interstices of which turbid serum exudes.

The surface of the entire lung on one side may be covered with this exudate even though only one lobe be consolidated. The changes in the lung are not necessarily lobar; indeed, the broncho-pneumonic type of lesions is more frequently associated than purely croupous inflammation.

If the disease has a chance to progress it terminates in empyema and as a rule, owing to the adhesions that develop, sacculated empyema results.

The majority of cases prove fatal at the height of the disease. In the first stage there is every evidence of an on-com-

ing pneumonia, together with severe pain in the side and the physical signs of pleurisy. Friction sounds are plainly heard and in the course of a few days distinct dulness, bronchial breathing and broncophony can be detected. These latter signs are somewhat obscured by the thick fibrinous layer, but never to the extent that an effusion would produce. Aspiration is negative, as a rule. An exact diagnosis is at times impossible, but the symptoms are too severe for a simple pleurisy and too indistinct for a pure pneumonia. When effusion develops, in left sided cases, the heart becomes displaced. Extension of the line of dulness beyond the mid-sternal line is also strong evidence of pleural effusion.

The prognosis is unfavorable as the pathological findings would naturally indicate. The younger the child, the worse the prognosis. Pericarditis is a common complication. Cases that survive must go through the course of an empyema with possibly severe crippling of the lung. When the process remains localized and abates in time, perfect recovery, barring some adhesions, is possible.

**Treatment.**—The treatment of croupous pneumonia is essentially the same as that recommended for *broncho-pneumonia*. Nevertheless there are certain remedies which are especially related to croupous exudations, in contradistinction to those of a purely catarrhal type, and they will, therefore, be called for here. Thus, *Ipecac* and *Tartar emetic* are less frequently indicated than *Bryonia* and *Sulphur*. In the early stages *Aconite* is by far the most useful drug.

*Iodine* is recommended by Kafka (*Homœopatische Therapie*) as being truly homœopathic to the croupous exudation, as well as to most of the symptoms. The *Iodide of Potash* he considers more valuable in apex pneumonia, especially when there is a tuberculous tendency.

The high fever and cerebral symptoms will call for *Belladonna* or *Veratrum viride*.

*Tartar emetic* and *Bryonia* hold the first place in *pleuro-pneumonia*.

Although *Phosphorus* is more useful in broncho-pneumonia than in croupous pneumonia, still it is of the greatest service where there is marked congestion indicated by dyspnœa; tightness across the upper portion of the chest; bloody expectoration; failing right heart and profound toxæmia.

*Sulphur* is one of the most useful absorbents in the *Materia Medica*, being especially useful in the third stage of pneumonia. It is recommended by Eidherr when exudation sets in, indicated by the appearance of the crepitant râle.

*Arsenicum* is well suited to those atypical cases of severe grade, in which the poison of influenza is added to that of pneumonia. In the presence of abundant bronchial secretion with dyspnœa and cardiac weakness, the *Iodide of Arsenic* is preferable.

Special symptoms are to be dealt with precisely as directed under *Broncho-pneumonia*.

#### PULMONARY TUBERCULOSIS.

Tuberculosis of the lungs during childhood manifests itself in a variety of forms, each depending upon the nature of the pathological findings for its clinical characteristics. Furthermore, it may be a primary or a secondary condition, and assume either an acute or a chronic course. The different varieties are: 1. Miliary Tuberculosis; 2. Caseous Pulmonary Tuberculosis; 3. Fibro-Caseous, or Chronic Pulmonary Tuberculosis. A fourth variety frequently encountered in adults, namely, fibroid tuberculosis of the lungs, is so rare during childhood that it need not be considered specially. Tuberculosis is separately discussed in the chapter on "Diathetic Diseases," to which the reader is referred for details regarding the factors concerned in the etiology of the disease as well as the bacteriology and pathology of the tuberculous infections.

**I. Miliary Tuberculosis.**—Diffuse miliary tuberculosis of the lungs may occur primarily, in which case it runs the course of general tuberculosis described as the "pulmonary

type" (see article upon "Tuberculosis"). In these cases the bacillus gains entrance into the lungs either through the bronchial glands or by means of the general circulation. In the latter instance the infection arises from a local focus in some other portion of the body, *e. g.*, a tuberculous joint affection. It may also be the terminal event of a chronic pulmonary tuberculosis, as a result of the discharge of the contents of a broken-down caseous mass into a bloodvessel, usually a branch of the pulmonary vein (WEIGERT). The form arising from bronchial gland infection is the type encountered during infancy. It is hardly probable that pulmonary infection through the lymphatic system from a primary tuberculous lesion in the intestines ever takes place.

II. **Caseous Pulmonary Tuberculosis**, also described as *acute* and *subacute pneumonic phthisis* ("galloping consumption"), is the form of pulmonary tuberculosis belonging to the period of childhood, in contradistinction to the infantile form described above. It is much more common than the chronic form, which, indeed, is rare in young children. Frequently it is engrafted upon a broncho-pneumonia, or occurs as a sequel to measles, whooping-cough or influenza. As a predisposing factor the tuberculous diathesis plays a most important rôle, no doubt more so than during infancy, when exposure to infection, either atmospheric or through the food, is liable to result in the development of the disease even in a healthy babe. Any illness capable of undermining the health and lowering the child's resisting power will also predispose to tuberculosis, even in the absence of a tuberculous family history.

The *pathological changes* in the lungs are either a diffuse pneumonic process which represents the lobar type and is rare, or a disseminated process representing the broncho-pneumonic type. This is the one usually encountered. We find isolated areas of consolidation, generally in the apical region, but not so strictly confined here as in adults. Usually both lungs are affected throughout, the bases sharing in the

pathological process. The consolidation is the result of the epithelial infiltration of the alveoli (*desquamative pneumonia*), and spreads from a terminal bronchus into the adjoining pulmonary parenchyma by contiguity of structure. Bronchitis and peri-bronchitis are associated with this process. The solid areas undergo caseation, which terminates in cavity formation if the case continue a sufficient length of time. Softening and excavation are the result of secondary infection with the streptococcus or staphylococcus (PRUDDEN). The fever accompanying this process is one of septic intoxication.

**Symptomatology.**—Tuberculous pneumonia begins with high fever, as an ordinary broncho-pneumonia, together with the development of signs of infiltration of the lung structure. Physical examination demonstrates areas of consolidation, usually the apices and bases. The percussion note loses its resonance and assumes a tympanitic quality over these areas, while auscultation reveals loud, moist and sonorous râles, accompanied by bronchial breathing.

The *temperature* range is high and remitting in character. As softening of the pneumonic deposits sets in and the vital powers fail, the temperature may fall to subnormal in the early morning hours, rising above  $102^{\circ}$  in the evening. Profuse sweating usually accompanies the fall in the temperature, and during the fever the skin is hot and dry and the cheeks flushed (*hectic fever*).

The *pulse* is weak and rapid, varying from 140 to 160 beats. Breathing becomes rapid and labored, often rising to 60 respirations per minute during the acme of the fever.

*Cough* remains troublesome throughout, at times being uncontrollable. Emaciation and anæmia develop rapidly, the child becoming pale and haggard, its countenance wearing an expression of great distress. Expectoration is usually scanty in the beginning, but toward the end it may become profuse, changing from mucus to muco-pus. Hæmoptysis may occur. The expectoration contains *Koch's bacillus*, and frequently also fibres of connective tissue, beside pus corpuscles and epithelial *débris*.

The *course* is rapid and fatal. Intermissions may occur, during which the disease remains quiescent for a short time, but it seldom fails to relight and terminate in a fatal issue. Instead of signs of resolution appearing at the end of a week or two, as in an ordinary broncho-pneumonia, or a crisis at the end of a week, as in a lobar pneumonia, the disease steadily progresses and the vital forces gradually fail. Death may occur within a period of two or three weeks from the beginning of the attack, or, owing to periods of temporary cessation of symptoms, be protracted beyond that time. A complete *arrest* of the process may take place, but it is seldom permanent, and, after several such remissions, the child succumbs in a few months. In general it may be said, however, that the course is slower throughout than that of an ordinary broncho-pneumonia.

*Gastro-intestinal disturbances* are present and hasten the decline. Diarrhoea is the most prominent of these.

The *circulation* gradually fails, and respiratory embarrassment advances. The extremities are cold, and enlarged capillaries may show prominently on the chest, even on the cheeks and hands, indicating pulmonary obstruction. A general oedema may set in toward the last, which usually disappears just prior to death.

Infection of the abdominal viscera may occur as a complication, especially if the case becomes protracted; a tuberculous meningitis may arise in like manner.

The *prognosis* is most unfavorable. It cannot be denied that occasionally, but very rarely, we encounter cases presenting every evidence of pneumonia of tuberculous origin that recover, or at least in which the disease is temporarily arrested. Even when evidence of a complicating meningitis is present this may occur. Such a case is reported by Baginsky (*Berlin. Klin. Wochenschr.*, 1881, No. 20), and I have personally seen cases that apparently presented this complication get well; but the prognosis must always be guarded. Fowler (Fowler and Goodlee "Diseases of the



Lungs,") expresses himself on this topic as follows: "The prognosis is in all cases unfavorable, but not so grave in the broncho-pneumonic as in the lobar form. In the less acute cases it may fairly be hoped that the disease pass into a sub-acute or chronic form."

**Diagnosis.**—A broncho-pneumonia in a child running a protracted course, giving no evidences of resolution, but rather those of destruction of lung-tissue, with hectic fever, should always arouse suspicion of tuberculosis. Likewise a lobar pneumonia running on without a crisis, but going into the above state, providing empyema be excluded, is of grave significance. This form, however, is rare, although I believe extensive consolidations are more commonly encountered in children than in adults. A clear family history of tuberculosis and the tuberculous diathesis, or a history of prolonged exposure to a tuberculous source of infection, offer strong presumptive evidence.

Positive evidence is offered by finding the bacillus of Koch in the sputum, with possibly fibres of elastic tissue. This diagnostic sign is, however, not always available, owing to the difficulty of obtaining sputum. An ingenious and most satisfactory method of obtaining the sputum for microscopic examination is carried out at Prof. L. Emmett Holt's clinics. A catheter or small stomach tube is inserted several inches into the œsophagus after a coughing spell, by means of which sufficient sputum can be obtained, as children invariably swallow their expectoration. This is a simple and perfectly reliable procedure and one that should never be neglected in suspicious cases. I am in the habit of attaching a glass syringe to the free end of the catheter for the purpose of aspirating enough expectoration for a satisfactory examination.

The character of the fever is in itself a strong evidence of the nature of the disease, and when taken in conjunction with the rapid emaciation and prostration, anæmia, diarrhœa, and sweats, the case becomes quite clear.

From this it will be seen that an ordinary broncho-pneumonia should not be confused with caseous pulmonary tuberculosis. A *diffuse broncho-pneumonia* attended by *acute dilatation of the bronchi*, however, may give rise to physical signs indistinguishable, for a time, from disseminated caseous tuberculosis, and we should therefore be cautious in giving a positive opinion (FOWLER).

The *physical signs* are those of either a disseminated broncho-pneumonia or of a lobar pneumonia. In the former, scattered areas of dulness, the note assuming a tympanitic quality, can be demonstrated, especially at the apices and the bases of the lungs, bilaterally distributed. The signs of bronchitis will be added, *i. e.*, large and small moist râles. The râles are at first bubbling in character, later assuming a crackling sound. Over the consolidated areas bronchial breathing may be elicited, rarely typical tubular breathing. Signs are not well marked, as a rule, on account of the large amount of secretion which clogs up the bronchi. In the early stages of pulmonary tuberculosis, physical signs may be characteristically scant and the fever be entirely disproportionate to the lesions demonstrable. Often the first signs are subcrepitant râles heard anteriorly in the mammary region, indicating invasion of the pulmonary parenchyma from the root of the lungs. (HOLT).

In the lobar form all the evidences of consolidation of an extensive area of lung-tissue will be found.

The *treatment* is that of pneumonia. When the fever runs high, cold sponge-baths every two to three hours are of decided benefit. Food should be given at regular intervals, and in the form of liquids or semi-solids of the highest nutritive value. Milk, eggnog, broths into which a raw egg has been stirred, or strained vegetable broth and raw-meat juice are most suitable. Alcoholic stimulation cannot be dispensed with; the average quantity will be about two drachms every 3 hours during periods of adynamia. It not only sustains the strength of the patient, but possesses some food value, and assists in controlling the cough.

A warm, moist atmosphere is to be maintained, together with the most thorough ventilation. The spraying of hydrogen dioxid about the room is advantageous. When the cough becomes tight and suffocative in character, a cold pack about the chest is of great benefit.

*Remedies* may be divided into two classes, namely, those calculated to affect the tuberculous process directly and those useful for special symptoms, such as cough, pyrexia, etc. To the first class belong notably the *Iodides*, especially the *Iodide of Arsenic*, and *Iodoform*, *Calc. carb.* and *phos.*, *Sulphur*, *Tuberculinum*. *Kreosote* is much used for its antiseptic action, but may do harm by upsetting the stomach. Remedies of the second class are *Chininum arsenicosum* and *Baptisia* for the pyrexia; *Silicea* and *Hyoscyamus* for the profuse sweats; *Apomorphia*, *Tartar emetic*, *Hyoscyamus*, *Phosphorus* and *Lycopodium* for the respiratory symptoms.

Some of these remedies combine, so to speak, both offices—for example, the *Iodide of Arsenic*. It is not only a constitutional remedy, but at the same time exerts a potent influence over the pyrexia and the catarrhal symptoms. Likewise, one of the *Calcareas* may fulfill every requirement if decided constitutional indications are present, the *Carbonate* suiting the fat, pot-bellied, scrofulous child best; while a poorly-developed, backward child, with flabby abdomen, lax joints and weak limbs, adenoid vegetations and enlarged tonsils, is more benefited by the *Phosphate*.

*Avian tuberculin* is recommended by Cartier for bronchopneumonia following one of the infectious fevers and assuming a "suspicious" type. The cough is incessant and tickling in character, the pulmonary symptoms become localized, emaciation sets in, and tuberculosis may be anticipated.

**III. Fibro-Caseous or Chronic Pulmonary Tuberculosis.**—The chronic form of pulmonary tuberculosis, in which fibrosis is added to the caseous process, is seldom encountered before the sixth year, not becoming a common disease until the time of puberty. No doubt most children showing a decided pre-

disposition to tuberculosis succumb to either the acute pulmonary form or to general tuberculosis before this period. Its course is identical with that of cases of consumption in young adults, in children above six years. Under this age it may be less typical, the regular hectic fever so characteristic in adults and the classical night-sweats being absent. Indeed, extensive destruction of pulmonary tissue may take place in association with a moderately high temperature without marked remissions or sweating.

A variety of lesions is found, the characteristic and most constant changes being caseation and fibrosis in conjunction with cavity formation. Owing to the tendency to destruction and excavation of pulmonary tissue, the term "ulcerative phthisis" is often applied to this disease. The coexistence of miliary granulations and areas of caseation and fibrosis indicates that the course has been marked by remissions, as well as periods during which the pathological process has been active. Such a period of activity often occurs immediately before the death of the patient, and during its continuance miliary tubercles in great number may form in parts of the lungs hitherto unaffected (FOWLER).

The seat of the primary lesion is one of the apices, and in the majority of cases the right. The process does not begin at the extreme apex of the lung, but about an inch below that point, and nearer the posterior and external than the anterior border, spreading thence backwards. The upper and posterior part of the lower lobe is involved often long before extensive infiltration or destruction of the upper lobe has taken place, and, as a rule, before the apex of the opposite lung is attacked. Infiltration of the lung at this site, together with infiltration of the apex, is almost positive proof of the existence of tuberculous disease of the lungs (FOWLER).

Associated lesions usually found are bronchitis, peri-bronchitis and bronchiectasis; emphysema (compensatory); pulmonary collapse, the result of bronchial obstruction; œdema and congestion at the bases; pleurisy, usually chronic fibrous,

although acute pleurisy with exudation is by no means an infrequent complication of phthisis. Lesions in other organs that may be encountered are tuberculous ulceration of the intestines, amyloid disease of the internal organs, tuberculous adenitis, meningitis and tuberculous arthritis.

Females seem more prone to consumption than males. The ages between twenty and thirty furnish the highest percentage of cases, the number gradually increasing from the fifth year to that time.

Certain previous diseases invite it. An attack of acute pleurisy often precedes the outbreak of pulmonary tuberculosis, or a lung impaired by a former pleurisy may become susceptible. Bronchitis may pave the way, but, according to Fowler, its importance is over-estimated. The same holds good for pneumonia.

Valvular disease of the heart bears an important relation to pulmonary tuberculosis. Congenital stenosis of the pulmonary orifice offers a strong predisposition. Mitral stenosis is not uncommonly found associated with consumption, an observation to which I can add my testimony. The antagonism between mitral disease, particularly regurgitation, and consumption, taught by Louis, is not absolute. Fowler has observed a number of cases in which the diseases co-existed, and others also have collected a sufficient number to disprove the theory.

Syphilis may predispose to tuberculosis by lowering the resisting power of the organism. It is even claimed, by Hochsinger, that both the virus of syphilis and tuberculosis may be transmitted to the offspring by the parent at the same time.

**Symptomatology.**—The only evidence of the disease to attract attention in the beginning may be *emaciation*, with gradually failing health. *Cough* is usually slight, and of a dry, hacking character, or there may be an associated bronchitis, with free expectoration. In some cases, recurring attacks of acute bronchitis precede the pulmonary involvement;

in others, infiltration of the lungs advances steadily in the absence of all catarrhal manifestations.

*Hæmoptysis* may be the first symptom to arouse suspicion. Even in young children it is frequently observed (BAGINSKY), usually auguring a rapid course. Hæmoptysis does not, however, always indicate destruction of pulmonary tissue; to the contrary, it is usually an early symptom, resulting from ob-



FIG. 39. ADVANCED CASE OF FIBRO-CASEOUS PULMONARY TUBERCULOSIS IN A BOY TEN YEARS OLD. NOTE EMACIATION, PARALYTIC CHEST, FLATTENING OF INTRA-CLAVICULAR SPACES, ALSO ADENOID FACIES

struction of some of the smaller blood-vessels by the tuberculous infiltration with resulting engorgement and rupture of the collateral vessels. Chest pains are due to either localized persistent.

*Physical examination* reveals an emaciated frame; long, flat chest, and superficial, feeble respiratory movement in



typical cases. The absence of the true paralytic thorax does not, however, exclude the possibility of pulmonary disease. When the process is active, the skin is dry and feverish. Commonly, enlarged superficial lymphatic glands can be felt in various regions of the body. The clavicle stands out prominently, as do also the angles of the scapulæ, and the infra-scapular region is flattened. Palpation reveals increased vocal fremitus in either one or both infra-clavicular regions; the percussion note is dull in the supra-clavicular region, and the area of dullness often extends down as far as the third rib anteriorly, occupying the interscapular space on one or both sides of the spinal column posteriorly. The dullness may be associated with a suggestion of tympanitic quality. Auscultation reveals, in the early stages, harsh breathing in the affected apex, associated with fine, crackling râles. Broncho-vesicular breathing soon develops. As infiltration advances, bronchial breathing can be elicited in the infra-clavicular space. The first place this can usually be demonstrated posteriorly is at a point opposite the fifth dorsal spine, midway between the border of the scapula and the spinous processes of the vertebræ (FOWLER). As softening and excavation occur, the signs of cavity are added.

*Fever* is an indication of the activity of the process. When not exceeding 100.4° F. it may be considered purely of tuberculous origin; when higher, it is due to secondary infection, and usually betrays its septic character by marked remissions (KOCII). While this is true in adults, it does not hold good in young children in whom the fever always tends to run high. Periods of latency may occur, during which there is no pyrexia, although the pulse, as a rule, is weak and rapid throughout the entire course of the disease. As characteristic of the tuberculous pulse, it is claimed that the number of beats per minute is not influenced by reclining or standing, as occurs normally.

The morning temperature is frequently subnormal, even during periods of quiescence. With infiltration and begin-



ning softening, the evening temperature rises to  $100^{\circ}$  to  $100.5^{\circ}$  F. Secondary infection and rapid disintegration of lung-tissue are accompanied by a higher evening rise, the fever assuming the hectic type. At times, extreme fluctuations in temperature occur without causing much distress to the patient. Fowler is of the opinion that high fever may be present without septic infection, simply indicating a rapid progress of the disease in an organism still capable of reaction. This, however, is at variance with the teaching of Koch and Prudden. Paroxysms of high fever, followed by sweating, invariably indicate an admixture of septic intoxication. *Night-sweats* are a common and most distressing symptom; ordinarily they simply indicate exhaustion, occurring as the temperature falls to normal or subnormal.

The *alimentary tract* becomes deranged, and anorexia and diarrhoea are common complications. The latter symptom, occurring at the termination of the disease, indicates intestinal ulceration. Vomiting may be a troublesome symptom, resulting either from severe coughing paroxysms or gastritis.

*Albuminuria* is more common in children than in adults (BAGINSKY).

In rapidly progressing cases a distressing cough, with free expectoration of yellowish, lumpy muco-pus containing the bacillus in large numbers, will be found. Hæmoptysis is—generally associated with such cases.

*Chronic fibroid phthisis* may be encountered in children, but it is rarer than the above variety. In these cases there is usually a dry, harassing cough and less pyrexia, while, pathologically, fibrosis is in excess of the infiltrative process. The course is slower than that of fibro-caseous tuberculosis, but in the majority of cases an acute tuberculous complication brings on a fatal termination (BAGINSKY).

The *prognosis* is unfavorable, especially when the disease develops at the period of puberty—a time when the organism requires every spark of vitality for its growth and development, and at which there is the strain of school life to be

considered. In girls, the tendency to chlorosis is also an unfavorable event. In younger children, if the course be not an acute one, the prognosis is more favorable, but still grave. Cases have no doubt been checked, but it is impossible to foretell a relapse or a later complication, such as meningitis, setting in. If arrest in the stage of infiltration can be accomplished, the prognosis is favorable. The constitution and family history must also be taken into consideration in forming an opinion as to prognosis. As Duckworth puts it, we do not cure our tuberculous patients; all that we can do is to place them under conditions favoring an arrest of the process.

A positive *diagnosis* is based upon a demonstration of the physical signs of infiltration and destruction of lung-tissue described above, the character of the fever, and the finding of the *bacillus of Koch*. A combination of any two of these data affords the strongest presumptive evidence of the existence of phthisis. Early in the disease, however, at which time it is most important that the malady be recognized, it is not always possible to find unmistakable evidence of tuberculosis; and especially in children are we at a great disadvantage, owing to the difficulty of obtaining sputum for microscopical examination. If the child cannot be made to expectorate into a cup, the stomach-tube should be passed as directed above (p. 301). Cough and emaciation in a child with a tuberculous family history, or with the history of having been exposed to such infection, together with slight evening pyrexia, are sufficient data to warrant a most thorough examination of the chest. The finding of a few localized subcrepitant râles at the apex of the lung, together with a prolonged expiratory sound in such a case, will enable us to make a diagnosis of beginning pulmonary tuberculosis. Later, as the classical symptoms of the disease develop, the diagnosis is comparatively easy. *Chronic purulent bronchitis* is, perhaps, the most frequent condition we are called upon to differentiate; but here the absence of the bacillus and the negative condition of the lungs will exclude tuberculosis.

**Treatment.**—In the treatment, prophylaxis is of first importance. Children presenting a tuberculous family history are liable to succumb to pulmonary tuberculosis on account of an inherited constitutional weakness. This predisposition is not, however, confined to such alone, as any constitutional enfeeblement in which the resistance of the organism is subnormal, especially when the chest is underdeveloped, offers a predisposing factor. Such children should be brought up in a locality where fresh air in abundance can be enjoyed, and they should be encouraged to lead an out-of-door life rather than be urged on in their studies. Particular stress should be laid on the physical development of the chest by suitable and methodically carried out breathing-exercises and calisthenics; and for overcoming the cold-catching tendency, a cold sponge-bath, followed by brisk rubbing with a coarse towel, is most efficacious.

A careful inspection of the nose and throat should be instituted early to determine the presence of local pathological conditions that may interfere with the proper performance of the function of respiration. The importance of early recognizing adenoid vegetations or enlarged tonsils, and promptly removing them by appropriate means, cannot be overestimated. And, lastly, it must be accepted as a fact beyond dispute that the most important prophylactic factor is the avoidance of giving entrance to the *bacillus of Koch* into the system. The infant's food should, therefore, be sterilized, unless it is positively known to be free from contamination. Nor must it be brought up in an environment menaced by the presence of a consumptive. The same holds good with older children. Until more rigorous sanitary measures are enforced and the consumptive is educated to dispose of his expectoration in a safe manner and avoid too intimate relations with those about him, the disease will not decrease very materially.

When the disease becomes established it behooves us to decide whether the patient is to be cared for at home or sent to a more suitable climate. It is worse than useless to send

away a patient whose condition is an acute one, or in whose lungs advanced destructive changes have already occurred, and pronounced emaciation, fever and night-sweats exist. On the other hand, a timely change of *climate* has saved many a life, especially if the patient can pursue an outdoor life. The requirements of a suitable climate are pure, uncontaminated air, equable temperature, and a maximum amount of sunshine. High altitude is by no means necessary; it best suits cases in which the disease is limited and there are no cavities. It may prove disadvantageous to some cases by bringing on dilatation of the air-vesicles on account of the rarified state of the air, thus making it impossible for the patient to return to a low region. Hæmoptysis also contra-indicates a high altitude, and neurotic temperaments are aggravated thereby. A moderate altitude is preferable in most cases. The most suitable locations offering this natural advantage are the Adirondacks, the Southern pine regions, and the great plains bordering the Rocky Mountains. A location at sea-level seems better for chronic cases with emphysema, especially when there is nervous irritability, insomnia and loss of appetite. It is also beneficial in septic pyrexias. Many consumptives do not mind cold weather; in fact, it benefits them. For such, Mt. Pocono, the Adirondacks and Denver, Col., are good locations. Others, again, especially those in whom there is considerable bronchitis, are required to seek a warm, moist climate, especially in winter. Florida, the coast region of Southern California, and the Bernudas offer these advantages. The main feature of climatic treatment, however, is the outdoor life invited thereby. No other form of treatment has yet given the promising results obtained in the sanatoria in which *open-air treatment* is systemically carried out, combined with forced feeding, hydro-therapy and judicious exercise.

When it is impossible to send the patient away from home, he should receive all the benefits of the *open-air treatment*. When he is able to be out, he should enjoy every hour of sun-

shine available. If he is too weak to walk, or if there is fever, he should sit in the sun, well protected with sufficient clothing and screened from draughts. In winter, as well as in summer, the windows of the sick-room should be kept open. During the night the sleeping-chamber must be kept thoroughly ventilated, there being less harm in night air than in a stuffy atmosphere.

The *diet* is very important. So long as the appetite remains good and diarrhoea is absent, the case should not be despaired of. A change of climate often brings about a restoration of appetite when that has been on the wane, and may in this way alone confer great benefit. It is important to feed the patient as much as he can take; in fact, overfeeding has even proven beneficial in some instances. Osler has seen good results following Débovè's method of introducing a mixture of milk, egg and finely-powdered meat into the stomach through a stomach-tube, three times daily, in cases in which gastric symptoms were distressing. Raw eggs are especially adapted as a food for the tuberculous. Cod-liver oil is usually well borne by children, and is useful so long as it does not disturb the digestion. Even in the presence of pyrexia not above 100.4° F. we should not refrain from liberal feeding. Alcoholics are useful here, particularly when they tend to increase the appetite. Eggnog is a desirable form in which whisky can be administered. *Raw meat* is supposed to possess antitoxic properties, and can be administered as balls of chopped meat rolled in pulverized sugar, in which form children will usually take it readily. Personally, I consider it one of the most valuable foods for the tuberculous.

*Special Symptoms.*—When there is continuous *pyrexia*, or high evening temperature, rest in bed is imperative. Sponging with tepid water to which alcohol has been added exerts a refreshing and tonic influence, besides being a safe means of reducing temperature. The so-called "antipyretics" are positively harmful. Such remedies as *China*, *Chininum arsenicum*, *Baptisia* and *Ferrum phos.* present special indications

for their selection in the pyrexia of tuberculosis, and exert a most favorable influence over the same. Full indications will be given later on.

*Cough.*—A cough which occurs in the morning and is accompanied by expectoration is useful, and should not be checked. Expectoration is materially aided by giving the patient a cup of hot milk, to which a teaspoonful of rum has been added, in the morning on awaking. On the other hand, a cough that continues during the night, causing loss of sleep, must be controlled (FOWLER).

The old school employ *Codein* for this purpose; but we have among our remedies most efficient means for controlling the cough, with which we do not run the risk of drying up secretions or overcoming reflex irritability to a dangerous degree. I would especially mention *Hepar sulph.*, 3x trit., as a most valuable remedy for the teasing night-cough of phthisis. *Drosera* is highly recommended by Hughes (*Manual of Therapeutics*) for cough depending upon increased reflex excitability. Beside these, *Hyoscyamus*, *Iachesis*, *Ipecac* and *Corallium rubrum* should be studied. When profuse expectoration is present *Stibium iodide* 2x (GOODNO), *Arsenicum jod.*, *Lycopodium*, *Stannum met.* and *Calc. carb.* are the remedies most likely to prove useful. They must be carefully differentiated in order to yield the best results.

*Hæmoptysis*, when slight and associated with tightness across the chest and hoarseness, calls for *Phosphorus*. Hughes places *Phosphorus* foremost when the air-passages are much implicated in the morbid process. *Geranium maculatum*, tincture, has proven of great benefit in profuse bloody expectoration.

The inhalation of *Creosote*, a few drops in a mixture of alcohol and chloroform, is often efficient in allaying an irritating cough and in improving the character of the expectoration when it becomes offensive.

*Night-sweats* are often uncontrollable, and try the physician's skill to the utmost. I cannot see the feasibility of



using extreme measures to check the same, as the sweating is only a sign of exhaustion when it occurs during sleep, or the natural termination of the febrile movement when it occurs at the decline of the fever. Our aim should be to build up the patient, and, if necessary, we may administer a stimulant at bedtime. When due to fever, a tepid or cold sponge-bath at bedtime is beneficial. *China* tincture is a good remedy in these cases owing to its tonic properties. *Silicea*, 6x trit., acts most satisfactorily when there is pulmonary disintegration. I have seen an *Iron* tonic gradually relieve the condition where the usual routine treatment had been used without success. Hughes recommends *Iodine* for nocturnal sweats. *Phosphoric acid* 3x will do a great deal for the debility resulting from sweats, diarrhoea and bronchorrhoea. *Jaborandi* is homœopathic to profuse sweating, and has given good results. Goodno recommends *Agaricin* ix, one grain at bedtime. *Atropin* is the standby of the old school.

*Diarrhœa*, when due to catarrh of the bowels, can be controlled by restricting the diet to semi-solids and selecting the proper remedy. *Phosphoric acid* is the most important one. When there is tuberculous ulceration of the bowel, slight hope for improvement is offered. This is the form encountered as a terminal stage of the disease. *Arsenic* may benefit this condition and should be tried.

*Gastric disorders* may result from overfeeding. The best evidence of this is the presence of undigested food-particles in the stools (FOWLER). When there is purely a gastric incompetency, *Nux vomica* proves of great value. A catarrhal condition calls for such remedies as *Pulsatilla*, *Hydrastis* and *Ipecac*. *Kreosote* is indicated when there is vomiting of glairy mucus, usually in the morning. It is a favorite remedy of the old school to improve the digestive function, increasing the appetite and checking flatulency.

*Laryngeal symptoms* supervening during the course of phthisis are mostly catarrhal in nature. *Spongia* is the chief remedy (HUGHES). Tuberculous laryngitis (ulcerative) requires the attention of a specialist.



The following list of remedies, with their clinical indications, may be studied for a fuller knowledge of the therapeutics of phthisis :

*Aconite*.—Pleuritic stitches, and blood-spitting after taking cold. *Ferrum phos.* is similar, but under this remedy there is less circulatory excitement, and anæmia and vasomotor disturbances are pronounced.

*Arsen. alb.*—Dyspnœa from exertion; cough between 1 A. M. and 3 A. M. Fever-heat and chilliness intermixed. Restlessness and thirst for small quantities of water. There is prostration and emaciation; anæmia and œdema of ankles; terminal diarrhœa. Mostly indicated in the *pneumonic type*. *Arsen. jod.*, 3x trit., freshly prepared is well suited to the *fibro-caseous form* of the disease when there is profuse purulent expectoration; emaciation; hectic fever and prostration. *Stibium iodide*, 2x trit., is highly recommended by Goodno in cases presenting profuse muco-purulent expectoration. *Stannum iodide* has profuse purulent expectoration easily raised, and of sweetish taste. It is more useful in chronic bronchitis.

*Baptisia*.—Chill in forenoon or afternoon, followed by heat and perspiration; general weakness and languor. *Baptisia* is one of the best remedies for the pyrexia of phthisis, and has been extensively used since it was first recommended by Dr. J. S. Mitchell. It is usually employed in the tincture and lower dilutions.

*Bryonia*.—Cutting pleuritic pain when taking a deep breath or coughing. Dry, deep cough, the irritation starting from the epigastric region.

*Calc. carb.*—"Pre-tubercular stage" in strumous subjects, the characteristic features being a form of indigestion associated with acid eructations and difficulty in assimilating fats (HUGHES). Pale, rapidly-growing youths (*Phos. acid*) or scrofulous children are especially benefited by this remedy. In the later stages it is indicated by tendency to perspire on slightest exertion; damp, cold feet; shortness of breath on ascending stairs; expectoration consisting of mucus with an

admixture of pus which sinks in water, leaving the frothy mucus floating above.

*Carbo veg.*—Flatulent dyspepsia and chronic hoarseness.

*China.*—Septic fever, consisting of a chill, followed by high fever and sweat, usually occurring at regular intervals. Anorexia ; chronic diarrhœa. (Tincture and lower dilutions.)

The *Arseniate of quinine*, 3x trit., is better indicated when the pyrexia is more irregular, especially if arsenic symptoms are present.

*Ferrum phos.*—Fever in the early stages, before septic infection has set in. Hæmoptysis in the early stages not dependent upon excavation of lung-structure.

*Hepar sulph.*, 3x trit., two grains every hour at night until cough is relieved. The cough is due to a persistent irritation in larynx, not relieved by free expectoration. It is excited by uncovering any part of the body, or by contact of body with cool bedclothes on first retiring. There is usually slight hoarseness, with rattling of mucus in larynx, but, as before stated, expectoration does not relieve the symptoms. *Drosera* has a deep, spasmodic cough presenting this element of hyperæsthesia, but there is not the free secretion present in *Hepar*. *Hyoscyamus* has symptoms of cough worse on lying down at night ; dry, spasmodic and titillating in character.

*Iodine.*—This remedy also presents characteristic cough symptoms. “Constant tickling in the windpipe and under the sternum, with expectoration of a transparent mucus, sometimes streaked with blood. Morbid hunger, even soon after a meal, and yet loss of flesh. Dark hair and eyes” (C. G. R.).

*Kali carb.*—Sharp stitches in chest ; cough worse 3 A. M.; puffiness of upper eyelids and swelling of ankles.

*Lachesis.*—Cough during sleep without awaking the patient ; chilliness, followed by fever, with great talkativeness ; sensation of suffocation ; fluttering of heart ; offensive stools.

*Lycop.*—Expectoration of large quantities of pus after neglected pneumonia (C. G. R.). Cough day and night, the ex-

pectoration tasting salty. Hectic fever, with circumscribed redness of cheeks, usually late in afternoon (four P. M. to eight P. M., aggravation of symptoms). During the fever we often observe automatic fan-like movements of the alæ nasi, not due to dyspnoea, but sympathetic with the pulmonary disturbance. "It suits cases of a chronic and passive character, and is, I think, especially useful when phthisis occurs in young men." (HUGHES.)

*Nux vom.*—Digestive derangements and aggravation of cough symptoms from overeating. *Kreosotum* 2x is one of the best remedies for persistent vomiting in phthisis.

*Phosphorus*.—Tormenting cough, often with hoarseness; worse toward midnight; tight and painful. There is tightness across upper portion of chest; inability to lie on left side. "Cough in the earlier stages of phthisis, with unusual implication of the air-passages in the morbid process." (HUGHES).

*Phosphoric acid*, acts restoratively when the system has been drained by long-continued diarrhoea or persistent night-sweats.

*Sulphur*.—Delayed resolution after pneumonia; chronic catarrhal deposits at apices, with a few moist râles. Neurasthenic individuals. Weak, gone feeling at 11 A. M., with craving for food or a stimulant. Vasomotor disturbances.

*Iodoform*, 3x trit.—Two one-grain tablets four times daily. It has given me most promising results in incipient cases of fibro-caseous pulmonary tuberculosis, and I use it in preference to the other iodides in the stage of infiltration.

*Tuberculin* (KOCH) has been successfully employed in broncho-pneumonia, and is considered by Arnulphy capable of stopping the progress of incipient cases of tuberculosis of the lungs in a large proportion of cases (*Clinique*, June, 1897). *Avian tuberculin* is recommended by Cartier for suspicious broncho-pneumonia. These nosodes have usually been given in the higher dilutions, either the 30th or 100th, although Mersch obtained his results from the 6th.

## EMPHYSEMA.

Overdistension of the air-vesicles of areas of pulmonary tissue occurs as a complication of almost any of the acute affections of the respiratory tract, resulting from either an interference with the function of a considerable portion of the lungs (*vicarious* or *inspiratory emphysema*), or from an obstruction higher up in the tract, leading to dilatation and even rupture of air-vesicles during expiration, especially when this is performed in a forcible manner. The latter variety, or *expiratory emphysema*, is by far the most pronounced form in which this condition is met with acutely, occurring as a common complication of whooping-cough, croup, asthma and measles, especially in rachitic children or those of lax fibre. It has also resulted from forcible expiratory efforts performed voluntarily, and from the inflation of the lungs of the new-born in cases of asphyxia. Chronic emphysema is occasionally seen in children as a result of chronic bronchitis and organic heart disease.

Anatomically, emphysema is classified as *vesicular* or *alveolar*, and *interstitial*. In the latter form there is an escape of air into the connective-tissue stroma of the lungs, sometimes burrowing beneath the pleura and along the mediastinum into the subcutaneous tissue of the supra-clavicular spaces. Only then can it be distinguished clinically from the vesicular form when it makes its appearance externally, in the above manner.

The *chronic* form, or *substantive emphysema*, is defined by Delafield as a chronic interstitial inflammation of the lungs in which the dilatation of the air-spaces is a secondary phenomenon. Accordingly, it is a condition whose etiology and pathology are analogous to that of chronic endocarditis, endarteritis and nephritis.

In acute emphysema the upper lobes are principally affected, and most markedly in their anterior borders. In the chronic form both lungs are more or less affected in their entirety, but seldom to the great extent observed in adults.

The *symptoms* of a compensatory emphysema are always obscured by the original disease. Hyper-resonance, bulging of the supra-clavicular space during the expulsive efforts of coughing, exaggerated vesicular murmur and dyspnoea are all suggestive.

Chronic emphysema presents the typical barrel-chest; feeble respiratory murmur with prolonged expiration; diminished area of cardiac dullness; cyanosis, dyspnoea, cough and expectoration; vesiculo-tympanitic percussion-note. It must be remembered, however, that none of these signs are as pronounced as in adults, and the younger the child, the less the aberration from the normal.

In both instances treatment is to be directed to the primary disease.

Such remedies as *Arsenicum*, *Arsenicum iodide*, *Aurum mur.*, *Ipecac*, *Lobelia* and *Grindelia* will be required for the symptoms of the disease *per se*. *Coca* and *Quebracho* are lauded by Hale as the only remedies giving continuous relief.

Constitutional remedies are valuable in rachitic children, notably the *Calcarea*, *Silicea*, *Ferrum phos.*, *Baryta carb.* and *iodide*, *Fluoric acid* and *Sulphur*.

#### PLEURISY AND EMPYEMA.

Inflammation of the pleura is rarely seen as a primary disease during childhood, but it is quite a common accompaniment of pneumonia, especially of severe forms of broncho-pneumonia. Pleurisy without exudation may accompany pulmonary disturbances of all kinds, and the frequency with which adhesions and thickening of the pleural membranes are encountered in the general run of autopsies upon children points to the great prevalence of this condition.

The exudative variety of pleurisy in children is almost invariably an empyema, and occurs most frequently as a complication of pneumonia, or develops simultaneously with the pneumonic process, which is the case in the *pleuro-pneumonia* described by some authors as a separate clinical condition. (See page 204).

The acute infectious fevers are responsible for the development of some cases of pleurisy, and in older children a purely serous effusion may occur as a result of tuberculosis or the rheumatic (?) diathesis.

The micro-organisms playing the most prominent rôle in the etiology of purulent pleurisy are the *pneumococcus*, the *pyogenic micrococci* and the *bacillus tuberculosis*. *Pneumococcus pleurisy* is the most frequent form. It may occur simultaneously with a pneumonia, or, what is more frequently the case, secondarily to the pulmonary affection, sometimes appearing several weeks later (STRAUSS). The exudate may be either sero-fibrinous or purulent. In the latter case the effusion is thick, creamy or greenish, and not clotted. The prognosis is better than in the other forms; the course is also milder.

*Streptococcus pleurisy* is more common in adults. The prognosis is not so favorable as in the pneumococcus variety. The course is more prolonged and the fluid re-accumulates after expiration.

*Tuberculous pleurisy* may occur primarily, that is, in the absence of pulmonary tuberculosis; but in these cases tuberculosis of the bronchial glands is generally present. The effusion is sero-fibrinous at first and gradually becomes purulent. The course is slow and unfavorable.

**Pathology.**—In the early stages of a pleurisy the membrane appears injected and lustreless; later, it becomes roughened and coated with a layer of fibrinous exudate. The extent of this process depends upon the severity of the attack, and it will vary from a delicate film of fibrin, coating only that portion of the pleura directly covering the affected portion of lung in a pneumonia, to a general involvement of the entire pleural cavity, with a thick layer of inflammatory products plus an abundance of sero-pus. In these pronounced cases the pleura appears coated with a yellowish-green deposit of varying thickness; the opposing surfaces may become adherent, forming pockets in which an abundance of pus is found.



If serum is poured out freely during the first stage, adhesions do not occur, at least not to a very great extent. This fluid soon becomes purulent from the free admixture of leucocytes.

**Symptomatology.**—An attack of pleurisy may be ushered in with repeated chills, as in adults, or with convulsions, which are especially common in infants. A dry, hacking cough and sharp, sticking pains in the side are the natural accompaniments of the inflammatory process. When free exudation takes place the pain disappears, but with this a new series of symptoms develop.

The pain is expressed by severe crying after each coughing paroxysm or when the child is moved; there is also a tendency to lie upon the affected side, together with increased abdominal breathing. If the child be old enough to express its suffering, it may mislead us by referring the pain to the epigastric region.

With the appearance of fluid, which is mostly of a purulent nature, dyspnea develops, its severity depending upon the amount of fluid present. The cough may become more and more severe, owing to a complicating bronchitis.

The fever is remitting in character, seldom very high, rarely running above  $103^{\circ}$  F. As the acute symptoms subside a slight afternoon rise may remain to indicate that the condition has become chronic, as it is very rare for an empyema to recover spontaneously. Obscure cases of sacculated empyema running a high fever for several weeks are occasionally encountered, and may prove very puzzling. Such a case, fluctuating between  $98^{\circ}$  and  $106.2^{\circ}$  F., has been recently reported by Holt (*Archives of Pediatrics*, Jan., 1902).

Cases of pleurisy, fully recovering within a short period of time, and without surgical treatment, have been either a dry pleurisy, a pleuro-pneumonia with scanty exudate, or a serous effusion into which no micro-organisms have gained entrance. An empyema resulting from infection with the pneumococcus may recover spontaneously in the course of two or three weeks, but those of streptococcus or tuberculous origin



seldom recover without surgical interference. The last mentioned is, indeed, rarely benefited by any form of treatment.

When pleurisy develops secondarily to another disease, its course is not essentially different from the above; thus, in a pneumonia there will be a post-critical rise in the temperature with all the attending symptoms of pleurisy and effusion, (Fig. 40). Sometimes, however, it is impossible to say just when the pleurisy has developed, the increasing dyspnoea, pain and cough indicating the addition of this serious com-

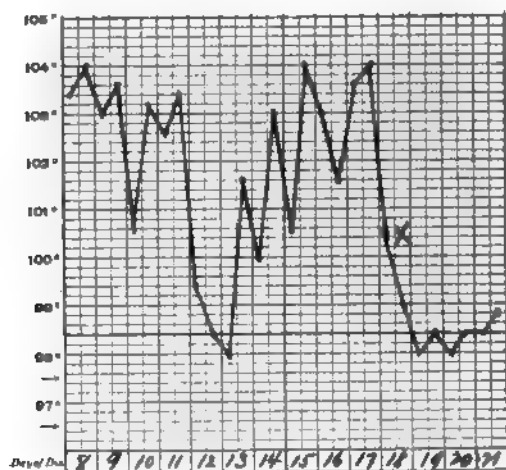


FIG. 40.—TEMPERATURE CHART FROM A CASE OF EMPYEMA DEVELOPING AFTER PNEUMONIA OF THE LEFT LOWER LOBE. A DRAINAGE TUBE WAS INSERTED AT (X) WITH RESULTING DECLINE IN THE FEVER.

plication. This frequently occurs in broncho-pneumonia, and as both conditions are then practically inseparable, the term *pleuro-pneumonia* has been rightly applied here.

The *physical signs* by which pleurisy is recognized in children are mainly those indicating the presence of fluid in the thoracic cavity, as the early signs, namely, the friction-sound and local tenderness, are not so readily elicited here as in adults. By observing the posture of the child, however, and

the fact that coughing produces severe pain, we often suspect a case in its early stages and are enabled to verify the diagnosis when the effusion appears. Conditions in which subcrepitant râles are present are a frequent source of error, they being easily mistaken for friction-sounds during infancy. For this reason the diagnosis of pleurisy depends upon a correct interpretation of painful inspiration, painful cough, the characteristic onset and fever, and, still later, the demonstration of a pleuritic exudate.

In the early stages of pleurisy fixation of the thorax from the pain is often observed in children, producing a voluntary scoliosis, as pointed out by Ziemssen. As a result of this abnormal position, the ribs are brought closely together on the affected side and the percussion note becomes dull. Under these circumstances, therefore, dulness may be observed before exudation has actually set in.

After exudation occurs the symptoms are more characteristic. If the amount of fluid be considerable, there will be a noticeable bulging of the chest on the affected side, together with diminished motion. When the fluid occupies the left pleural cavity the heart is displaced to the right; when occupying the right pleural cavity there is a downward displacement of the liver. The pleural fold is also displaced beyond the midsternal line.

Vocal fremitus is absent over the site of the fluid, while the percussion-note is flat and there is increased resistance. These two signs are among the most important data in the diagnosis of effusion. Percussing with the flat hand directly over the site of the fluid gives a very good demonstration of the resistance present. Above the level of the fluid tympanitic resonance is obtained when the lungs are not entirely deprived of air. The line of flatness will change its direction with a change in the position of the patient, providing the fluid is not inclosed by inflammatory adhesions.

In fresh cases bronchial breathing is very frequently heard above the line of dulness, which only gradually gives place

to the entire disappearance of the respiratory murmur with the increase in the exudate, (HENOCH, "*Vorlesung über Kinderkrankh.*") Rosenbach (*Nothnagel's Encyclopædia*, 1902) has not met with this peculiar type of breathing and he calls attention to the lack of specific signs in the pleurisies of children. The fact is, there are no constant signs. As large effusions are rare, the symptoms are less uniform than in adults. All authorities agree on the importance of the free use of the exploring needle. Even in moderately large effusions it is common to hear bronchial breathing and bronchophony over the entire back on the affected side. Moist râles may also be present to cause confusion.

In children under three years the fluid is usually purulent, and even until puberty this tendency prevails. According to Baccelli a purulent exudate is less likely to transmit the whispered voice, but this is not always the case. Subcutaneous œdema of the thorax on the affected side is not so commonly present in children as in adolescents and adults to indicate the purulent nature of the exudate. A positive diagnosis cannot, however, be made without the use of the aspirating needle, which is perfectly safe when used under proper aseptic precautions. In old cases, where the pus is too thick to be drawn into the needle, even this method will lead to error unless the negative result is properly interpreted. In a serous exudate, the presence of chain cocci, staphylococci, or the diplococcus pneumoniae, indicates that it will become purulent.—KOPLIK. Tuberculous pleurisy is recognized by finding the tubercle bacillus in the effusion and according to Dieulafoy by the exclusive presence of lymphocytes and red blood corpuscles. In the other forms of infectious pleurisy polynuclear and large mononuclear leucocytes predominate.

**Diagnosis.**—The early diagnosis of fluid in the chest is of the utmost importance, particularly as the recovery of the patient depends much upon the time when proper treatment has been instituted. Many difficulties may be encountered

in deciding upon this point, especially as the effusion is not generally a large one and because it is usually secondary to pneumonia—metapneumonic pleurisy. The history is therefore not as clear as in primary pleurisy. Again, owing to the strong tendency for the fluid to become encapsulated, it does not produce the characteristic physical signs expected of free fluid in the chest. The determination of the character of the fluid has been fully discussed above.

The chief indications upon which the diagnosis can be made are absence of vocal fremitus; flat quality of the percussion-note and resistance; bronchial breathing and broncophony over the entire affected side posteriorly and displacement of viscera. Koplik lays special stress upon displacement of the pleural fold. Normally these folds meet in the midsternal line and when there is considerable fluid in either side of the chest cavity dullness will be found to extend beyond the median line over toward the well side. In smaller effusions auscultatory signs are not characteristic and may be misleading on account of the good conduction of sound in the child's chest.

Empyema should always be suspected when the temperature remains high for a period beyond two weeks in cases of pneumonia, especially when bronchial breathing can be heard over an entire side.

Encapsulated fluid in unusual sites, such as the upper portion of the chest, is very difficult to differentiate from persistent broncho-pneumonia and abscess of the lung. In the latter condition percussion and auscultation give practically the same signs, but the presence of loud, coarse pleuritic friction sounds are of importance as favoring the diagnosis of abscess (HOLT, *Archives of Pediatrics*, Jan., 1904).

Pericardial effusion must also be borne in mind as a possible condition likely to be confused with sacculated empyema.

**Prognosis.**—Serous effusions are usually absorbed readily, seldom persisting over three weeks. If however pus producing micro-organisms gain entrance into the pleural cavity

the prognosis is immediately altered. As stated above, an empyema due to the pneumococcus presents the most favorable prognosis, although it may run a prolonged and tedious course, the usual period being from some weeks to two months (STRAUSS). This is the variety that may recover spontaneously, or after one or two aspirations. When the streptococcus is present the fluid tends to re-accumulate unless open drainage be instituted. The tuberculous variety is the least favorable. Spontaneous evacuation through the chest wall (usually in the region of the fourth or fifth rib) or through the bronchial tubes, by perforation into the lung parenchyma, is a not infrequent termination of the pneumococcus variety. The other varieties, however, rarely evacuate themselves and do not tend to reabsorb. At times perforation into the peritoneal cavity takes place with a fatal issue. The usual cause of death in an untreated empyema is the gradual exhaustion or amyloid degeneration accompanying prolonged suppuration. Tuberculosis is also liable to supervene.

When the fluid is removed early there is a fair chance for the compressed lung being restored to complete function; on the other hand, if the condition has been one of long standing, dense bands of adhesions have generally been formed to such an extent as to allow of but a partial inflation of the lungs, resulting in permanent deformity of the chest and spine.

**Treatment.**—Local treatment is of little avail in children, with the exception of the judicious use of hot applications and a flannel binder in the early and painful stage. Fluid which is present in considerable amount should be promptly evacuated if absorption is not progressing rapidly; under no circumstances should accumulations of fluid be allowed to remain in the chest for a period exceeding two weeks, unless decided improvement is noted daily. As the accumulations are almost invariably purulent in character, they are difficult to absorb.

Sometimes a partial removal of the fluid by aspiration produces sufficient relief of the intrapleural tension to excite the activity of the absorbents and lead to a complete recovery. By this method undoubted cases of empyema have been cured without open drainage. — (GOODNO.)

In cases of long standing, however, and in serous pleurisy of large effusion, displaying a tendency to rapid recurrence after aspiration and producing alarming pressure symptoms, open drainage is to be instituted. *Simple incision*, when practiced under the strictest antiseptic precautions, yields such prompt and lasting results that it has to a great extent superseded the operation for the *resection of a rib*. One of the advantages which this operation offers is the foregoing of the use of a general anæsthetic, the local use of Ethyl chloride or Cocaine being all-sufficient.

*Paracentesis of the thorax* is accomplished with either an aspirator, such as the Dieulafoy or Potain aspirator, or by means of a small trocar. Before inserting the trocar an exploratory puncture with a large size hypodermic needle should be made to locate the fluid. Negative results with the hypodermic needle do not, however, exclude the presence of fluid, as the puncture may not have been sufficiently deep, the pus may be too thick to flow, or the needle may become clogged with fibrin, preventing the entrance of the fluid. The usual site of puncture is the sixth or seventh intercostal space in the mid-axillary line or the seventh or eighth interspace posteriorly. The needle should not be inserted too close to the spine, and should be directed toward the upper border of the rib rather than to its lower, on account of the intercostal arteries. Koplik insists on puncturing at the site indicating fluid, as elicited by flatness and absence of vocal fremitus; when the empyema is localized this rule is absolutely essential to follow.

Having decided upon the best site for the puncture, the area is thoroughly cleansed and the trocar, previously sterilized, is forced through the thoracic wall with a slight rotary

movement. The thumb is firmly held at a distance of about one inch from the point and the trocar inserted to this depth if abundant fluid is present; the stylet is then withdrawn from the canula and the fluid allowed to flow into a pus basin. Care must be taken not to allow the fluid to run out too rapidly, as syncope may result therefrom. Coughing is excited by the operation, but this facilitates the expulsion of the fluid. As the child inspires it is well to place the finger over the opening of the canula to prevent air entering the pleural cavity. Should this take place it will, however, do no harm.

*Technique of Incision.*—The child is laid on its well side and the arm of the affected side held up by an assistant, thereby exposing the lateral region of the chest to its full extent. Beginning first behind the mid-axillary line, an incision is made in the sixth or seventh intercostal space and carried forward for a distance of one and one-half inches. The skin should previously have been scrubbed with soap and water and subjected to the action of a wet 1–2,000 Bichloride dressing for one hour. Ethyl chloride is the only anæsthetic required.

After dividing the skin and intercostal muscles an artery forceps is plunged through the pleura and an opening made sufficiently large to receive a drainage tube. The drainage tube is introduced with a tissue forceps for a distance of about two inches and its free end transfixed with a sterilized safety-pin. After all of the pus has been evacuated a dressing of sterilized gauze covered with absorbent cotton is applied, which will have to be changed once or twice daily, according to the amount of exudation forming. The tube should be removed every one to two days and thoroughly cleansed; as the case improves a smaller tube may be used until it is proper to allow the wound to heal. Irrigation of the pleural cavity is seldom necessary and is associated with a certain amount of danger. When a good-sized drainage tube cannot be introduced equally good results may be obtained from two smaller ones placed side by side.



Relapses may, however, occur on the removal of the drainage tubes, if there be a virulent streptococcus infection, necessitating the resection of a rib; and if the lung has become markedly crippled, leaving an open cavity in spite of complete recession of the affected side of the thorax, the operation of Estlander is to be considered.

In the tuberculous variety the ordinary operation for empyema seldom accomplishes anything, owing to the rigidity of the chest-walls and the complete collapse of the lung. Re-accumulation of fluid always occurs after aspiration, although more slowly than in a streptococcus infection. It may be said that, as a rule, non-interference is the best plan, unless the necessity be urgent (FOWLER).

**Remedies.**—*Aconite*, *Arnica*, *Belladonna*, *Bryonia*, *Kali carb.*, *Rhus tox.*, *Scilla* and *Tartar emetic* will be found useful for the early symptoms, they having a special relation to the inflammatory stage.

When exudation is abundant, *Apis*, *Arsenicum*, *Cantharis*, *Kali hydrojod.* and *Sulphur* are most frequently indicated.

In purulent collections one of the constitutional remedies, prescribed upon the temperamental and diathetic peculiarities of the patient, will yield most gratifying results and greatly hasten the progress of the case. *Ars.*, *Ars. iod.*, *Calc. carb.* and *phos.*, *Hepar*, *Iodium*, *Mercurius*, *Silicea* and *Sulphur* stand prominently among these.

*Acon.*—Sharp, stitching pain in side; high fever, restlessness and chills; after exposure to cold, dry winds or checked perspiration.

*Apis.*—Pleuritic effusion; scanty urine.

*Arnica.*—Traumatic cases; hæmorrhagic effusion.

*Arsenicum.*—Profuse serous effusion; dyspnoea; cachexia; prostration; empyema. The *Iodide of Arsenic* is well suited to tuberculous cases, as is also *Iodoform*.

*Asch. pias tuberosa.*—Sharp, stitching pains in the side; dry, hacking cough. Complicating pneumonia and tuberculosis.

*Bellad.*—Cerebral symptoms; complicating the infectious fevers or exanthemata.

*Bryonia.*—Early stage of all pleurisies, and in dry pleurisy frequently to the end. *Sulphur* is needed in the latter cases to complete the cure. Sharp, stitching pains, aggravated by motion and deep breathing; friction sounds and local tenderness.

*Calc. c.*—To absorb the pleuritic exudate. Scrofulous and rachitic diathesis.

*Canth.*—Profuse serous exudation; frequent cough; dyspnoea; palpitation; profuse sweats; great weakness; tendency to syncope; scanty and albuminous urine.—(E. FAIVRE.)

*Colchicum.*—Rheumatic diathesis; sour-smelling sweats; scanty, red, turbid urine, with abundant uric acid and some albumin.

*Hepar.*—Purulent accumulations; also dry, croupous exudate; abscess of lungs; hectic fever. "*Hepar* will often help to clear up the confirmed cases of purulent pleurisy where galloping consumption is apparently threatening."—(FISCHER.)

*Kali carb.*—Violent stitching pains, especially on left side, worse in early morning (after fresh adhesions have formed during sleep), accompanied by dry cough and palpitation of the heart. When *Bryonia* fails to give relief.

*Kali hydr.*—Serous exudations.

*Mercurius.*—Syphilitic or rheumatic diathesis; pains persisting after the fever subsides; constant chilliness, with tendency to sweat; gastro-intestinal catarrh; perihepatitis. *Merc. corr.* is useful in pleuritic effusions accompanying parenchymatous nephritis.

*Phosphorus.*—Complicating broncho-pneumonia. Pain in mid-sternal region and on both sides, especially when coughing. Also in empyema with Bright's disease; hypertrophy of right heart; amyloid changes. Hard, dry, distressing cough with hoarseness.

*Rhus tox.*—Acute rheumatic cases, after exposure to wet

or after physical overexertion. General aching and prostration; typhoid state.

*Scilla*.—Sharp stitching pains in side with broncho-pneumonia; prostration; cardiac weakness. Cannot lie on left side.

*Sulphur*.—Later stages of dry pleurisy and after the effusion makes its appearance in the exudative variety. *Sulphur* is a most valuable absorbent, and we are always obliged to come back to this remedy when others fail to improve the condition, or when clear indications for others are not present.

## CHAPTER XII.

### DISEASES OF THE HEART AND ITS MEMBRANES.

The heart affections of childhood are both congenital and acquired. Congenital affections may be either the result of foetal endocarditis or developmental defects and abnormalities. Acquired heart disease presents the same pathological phenomena observed in adult life, with, however, such clinical deviations from the adult type of a given disease as must necessarily result from the physiological peculiarities of the circulatory apparatus distinctive of child-life. Functional disorders are also encountered, but with greater rarity than in adult life, as the common causes for this train of symptoms, viz., abuse of coffee, tea, tobacco and alcoholics, also neurasthenia and hysteria, are infrequently active at this age. Reflex irritation, however, is a frequent source of cardiac symptoms in the child, notably, gastro-intestinal irritation, helminthiasis and teething.

The heart is relatively larger in infancy than in later life, but it does not increase in size proportionally with the growth of the child, developing only slightly during the first five years of childhood (Barthez and Rilliet). It occupies a higher and more horizontal position than in the adult, and for this reason cardiac dulness extends relatively further both to the right and to the left of the sternum. (Fig. 41.) Up to the sixth year dulness may extend beyond the right border of the sternum, and the apex is generally found outside of the left nipple-line up to the fourth year. Again, the apex may be in the fourth intercostal space until the sixth or seventh year. After the seventh year, however, it should be located well within the left nipple-line and in the fifth intercostal space, to indicate a perfectly normal condition. It is important to remember that the nipple is not invariably a fixed point, as it may

be found in the third or fourth intercostal space or over the fourth rib. Most frequently it is situated over the fourth rib, somewhat nearer the median than to the mid-axillary line.

Deep cardiac dulness extends beyond the left mammary line up to the second intercostal space, and on the right it

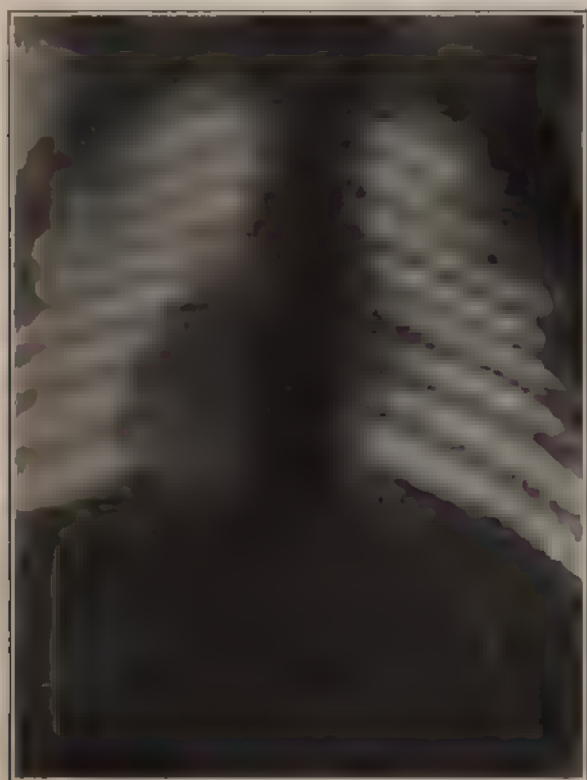


FIG. 41.—SKIAGRAPH OF CHILD'S CHEST, THREE YEARS OLD, POSTERIOR ASPECT, THE SHADED PORTIONS INDICATING THE HEART AND THE LIVER.

may reach to or even beyond the parasternal line in young children. (Sahli, *Topographische Percussion im Kindesalter*.) The same author has found this puerile type of heart as late as the twelfth year, although by the sixth year the rela-

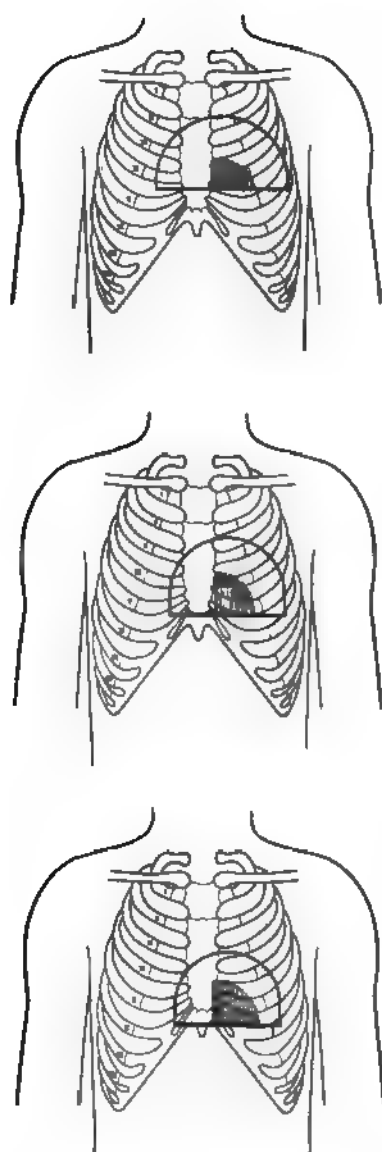


FIG. 42 —CARDIAC DULNESS AT ONE YEAR, SIX YEARS AND TWELVE YEARS.

tions as found in the adult are usually established. He further states that the area covered by the deep dulness differs in size and shape from the heart itself, owing to the projection of the borders of the heart upon a chest wall more convex than in the adult, and the admixture of a certain amount of "lateral dulness," resulting from the great resiliency of the chest wall. The heart is therefore not as large as the boundary obtained by deep percussion would indicate (Fig. 42).

Owing to the yielding character of the sternum and the costal cartilages, enlargement of the heart may cause a decided bulging of the front of the thorax up to the third year. This is usually seen in congenital heart disease. The third piece of the sternum may be displaced at even a later period, as Rotch points out, owing to the fact that it is ossified later than the upper portions. Pericardial effusion will cause bulging over the heart at any period of childhood.

The distance of the apex from the mid-sternal line I

have found to be from 4.5 cm. to 5 cm. in the new-born. By the tenth year it is 7 cm. in the average case. In a male child from one to two years old it is from 5.5 to 6 cm., usually a trifle less in females. From the fourth to the sixth year it averages 6 to 6.5 cm. and may reach 7 cm. by the seventh year. The yearly gain in the distance of the apex from the median line seems trifling and does not appear to correspond with the increase in size of the heart, but it must be remembered that the heart is relatively large in early childhood and also that it assumes a more vertical position with the fuller development of the child.

The *pulse* is soft and dicrotic in character during childhood. It is rapid and arrhythmic in infants. Its rate is about 130 at birth; 120 at end of first year, and usually remains about 100 up the fifth year.

The child's heart exhibits a greater resistance to organic disease than the adult's. The explanation of this is, according to Soltman ("*Der Kinderarzt*," ix., No. 2, 1898), the abnormal elasticity of the great vessels, the relatively greater muscular development of the heart, and the relation of the ventricles to one another. Intra-ventricular pressure has long been held to exert a pronounced influence in the development of endocarditis and its deformities, as shown by the overwhelming frequency with which it is found in the left heart after birth, and in the right during intra-uterine life.

The *blood pressure* in the arteries is considerably lower in the child than in the adult, owing to the relatively greater development of the aorta and the low arterial tension. The normal blood pressure in children up to the third year is about 90 to 109 mm. of mercury with the Riva Rocci sphygmomanometer while in adults it ranges from 125 to 135 mm. The volume of the heart compared with that of the aorta is almost three times as great at puberty as it is in infancy.

Normally the first sound heard at the apex is the loudest; next comes the pulmonary second best heard in the second interspace at the margin of the sternum. The aortic second



is heard best at the right border of the sternum, a little higher up. At puberty it is usually a trifle louder than the pulmonary second, although they may be of about the same intensity. It is wrong to speak of the pulmonary second sound being accentuated simply because it is louder than the aortic. As Cabot says, it must be distinctly louder than under normal circumstances in order to be of pathological significance.

The characteristics of cardiac murmurs (excluding those due to congenital defects) are summarized by Soltman as follows :

*Anæmic murmurs* are rare in the first four years, and even up to the eighth year, but they are comparatively common at puberty, at which time anæmia and chlorosis are prevalent. The low blood-pressure in the ventricles and large source of origin of the great blood-vessels in early childhood explains their infrequency, their reverse condition obtaining at puberty. They are heard loudest at the pulmonary valve, and are systolic. There must be no heaving impulse, accentuated second sound, or extension of the apex-beat beyond the mammillary line. The pulmonary area is so frequently the seat of murmurs that Balfour has referred to it as the area of auscultatory romance. It is well always to bear this in mind before venturing an opinion as to the existence of heart disease on so slender a basis.

*Cardio-pulmonary murmurs* (HOCHSINGER) are produced by the transmission of the contractions of the heart and its movements to the lungs. These murmurs are also systolic, and are differentiated from anæmic murmurs by their definite relation to the respiratory function, being increased during forced and suspended by a cessation of respiration. They are common in children with deformed chests, due to rickets or Pott's disease, and are best heard over the præcordial region.

*Endocarditic systolic murmurs* are heard in mitral insufficiency ; and for a long time this sign, together with a heaving impulse, may be the only symptoms of endocarditis, cardiac enlargement, accentuated second sound and increased tension in the pulmonary artery being absent.

Other murmurs which may be heard in chronic valvular disease are the *presystolic*, which may likewise be felt as a thrill running up into the cardiac systole. Occasionally a presystolic murmur may be heard where at the autopsy no mitral stenosis has existed, the valves simply being distorted or one of the chordæ ruptured. Again, mitral stenosis is not always accompanied by the presystolic murmur, but in such cases the murmur can often be brought out by causing the patient to exert himself. On the whole, the presystolic is the most fugitive murmur encountered.

The *Flint murmur*—a presystolic apical murmur heard in aortic insufficiency—is so rarely encountered in children that it need scarcely be considered. A *diastolic mitral* murmur may occur in old mitral cases where there is much dilatation without being indicative of aortic regurgitation. It is accompanied by a loud, banging, pulmonary second sound, and is explained by Steell as resulting from high pressure in the pulmonary artery. As the aortic diastolic murmur is sometimes heard at the apex, considerable confusion in diagnosis may arise, especially if we only take the murmur into consideration. The murmur Steell refers to should not be confused with the diastolic shock and reduplication heard in adherent pericardium. On account of the rapidity of the heart's action it is practically impossible to distinguish between a presystolic and a diastolic murmur.

#### CONGENITAL DISEASES AND DEFORMITIES.

As has been said above, congenital heart affections result from either foetal endocarditis or interrupted or abnormal development. Frequently, however, both of these processes act together—a mechanical obstruction in the circulation, as a result of endocarditis, leading to non-closure of the auricular and ventricular septa or of the ductus arteriosus. For this reason it is more common to find a combination of defects rather than an uncomplicated lesion. Thus, Holt found, from an analysis of 242 cases, that the most frequent lesions were a

combination of pulmonic stenosis with defective ventricular septum, pulmonic stenosis with defective auricular septum, the three lesions associated, or the first two with a patent ductus arteriosus.

*Fœtal Endocarditis.*—Inflammation of the endocardium in the fœtus is of the chronic or sclerotic variety, verrucose endocarditis being very rare (OSLER, *Keating's Cyclopædia*). Small, nodular bodies, the remains of fœtal structure (BERNAYS), and small, rounded, bead-like bodies of a deep purple color, which are the remains of a hæmorrhage (OSLER), have frequently been mistaken as evidences of endocarditis, leading to a misconception as to the prevalence of this affection. The characteristics of fœtal endocarditis are thickening of the segments of the valves, their edges becoming rounded and shrunken. The semilunar valves become obliterated, leaving a stiff, contracted ridge at the orifice of the great vessels. The right heart is most liable to endocarditis, as well as to errors of development.

*Congenital Anomalies.*—Mentioned in the order of their frequency, according to Holt, congenital anomalies of the heart may be classified as follows:

1. Defect in the Ventricular Septum.
2. Defect in the Auricular Septum, or Patent Foramen  
Ovale.
3. Pulmonic Stenosis, or Atresia.
4. Patent Ductus Arteriosus.
5. Abnormalities in the Origin of the Great Vessels.
6. Pulmonic Insufficiency.
7. Tricuspid Insufficiency.
8. Tricuspid Stenosis, or Atresia.
9. Mitral Insufficiency.
10. Mitral Stenosis, or Atresia.
11. Aortic Insufficiency.
12. Aortic Stenosis, or Atresia.
13. Transposition of the Heart.
14. Ectocardia.

*Defect of the ventricular septum* is most frequently associated with pulmonic stenosis or defect of the auricular septum. The defect is most frequently found in the anterior muscular portion of the septum (ROKITANSKY). If compensatory hypertrophy of the right ventricle supervenes, no apparent symptoms may be present. Cyanosis results from an obstructed venous circulation, with embarrassed respiration, cyanosis and cedema. This, and not the mixing of arterial with venous blood, is the cause of the cyanosis (BAGINSKY).

*Patency of the foramen ovale* may exist without any evidence of cardiac disease. When, however, other anomalies increasing the pressure in the right auricle co-exist, a mixing of venous and arterial blood takes place, with resulting cyanosis. Under these circumstances the child is liable to an early death.

*Stenosis of the pulmonary artery* is one of the commonest of congenital heart affections, as a rule being responsible for the existence of the above-mentioned anomalies. The usual cause for the stenosis is endocarditis, although there may be a developmental defect of the pulmonary artery (ostium) or of the conus arteriosus. The symptoms depend upon the amount of constriction at the pulmonary orifice. The infant may die shortly after birth with intense cyanosis and asphyxia, or it may grow up to adult life, with, however, signs of deficient aeration of the blood, cyanosis from undue physical exertion, coldness of the extremities, clubbing of the finger-nails, and mental and physical apathy. Simple pulmonary stenosis is found only before the thirteenth month according to Rokitansky. The obstruction to the circulation in the great majority of cases that do not die in early infancy leads to the defects mentioned above.

*Patent ductus arteriosus* does not necessarily produce symptoms. Hirst finds a certain degree of patency of the duct quite common in children during the first year of life. But in these cases there is no appreciable deviation from the normal circulation. The symptoms produced are hypertrophy and

dilatation of the right ventricle; dilatation of the pulmonary artery; dyspnoea and cyanosis; bronchitis. The physical signs are pronounced.

*Abnormalities in the origin of the great vessels* are rare, and lead to early death or make extra-uterine life impossible, unless there is an open foramen ovale or a communication between the pulmonary veins and the right side of the heart.

*Tricuspid insufficiency and stenosis* are grave defects, resulting from endocarditis. There may be complete atresia of the orifice, in which case a degree of circulation is maintained through an incomplete ventricular septum. The right heart becomes dilated and hypertrophied; there is cyanosis and tendency to venous hæmorrhages.

Affections of the *left heart* are rarer than those of the right, and result likewise from endocarditis. The symptoms and physical signs are the same as observed later in life.

The *symptoms* of congenital heart affections may be summed up as the indications of deficient aeration of the blood or a mixing of venous with arterial blood, and interference in the systemic circulation. Cyanosis is the most persistent symptom, and is, in fact, pathognomonic of congenital heart disease in the absence of other causes capable of exciting this phenomenon. Among these may be mentioned pneumonia, asphyxia, bronchitis, atelectasis, congenital pleurisy, partial occlusion of the trachea, degeneration of the blood, interference with the nerves of respiration.—(HIRST.)

Dyspnoea is another prominent symptom. Among the later manifestations of congenital heart disease are clubbing of the finger-nails, cold extremities, mental and physical apathy, deformity of the chest from hypertrophy, and dilatation of the heart. Hypertrophy will produce deformity of the sternum up to the third year.

The first symptoms are usually noticeable at birth, the child being a so-called "blue-baby." At other times they are very mild, and are only noticed when the child becomes excited or attempts physical exertion. Again, the defect may

not be suspected until an acute affection of the respiratory tract precipitates the symptoms, or it may not become apparent until the child grows up.

The *diagnosis* rests upon a recognition of the above-mentioned symptoms, together with the physical signs. According to Sansom, a *patent foramen ovale* is to be recognized by cyanosis without a heart murmur (in which case we must necessarily exclude all other causes for cyanosis), or by cyanosis with systolic and presystolic murmurs over the cartilages of the third and fourth ribs. The same observer also claims that *defective ventricular septum* is to be recognized by a loud systolic murmur over the præcordium and between the shoulders, not transmitted to the vessels.

In *tricuspid stenosis and insufficiency* there is hypertrophy and dilatation of the right heart, labored heart's action, præcordial thrill, loud systolic and diastolic murmurs at the apex.

*Stenosis of the pulmonary artery* presents a hypertrophied right heart; loud systolic murmur over the second and third costal cartilages to the left of the sternum, not transmitted into the carotids, and præcordial thrill. The pulmonary second sound is weakened. When these signs are present in a child over thirteen months old it can be taken for granted that there is an open foramen ovale. When the murmur is also transmitted into the carotids it points to associated septum defect. When there is a loud, buzzing murmur transmitted into the carotids and subclavians, together with accentuated pulmonary second sound and hypertrophy of both ventricles, there is probably associated an open ductus arteriosus (HOCHSINGER, *Auscultation des Kindlichen Herzens*).

*Patency of the ductus arteriosus* leads to rapid hypertrophy of the right ventricle and dilatation of the pulmonary artery, increased area of cardiac dulness, long-continued systolic murmur with thrill and cold surface. The presence of a thrill and a distinctly-defined area of dulness in the second intercostal space to the left of the sternum, above the heart, is of great diagnostic import (KOPLIK, *Diseases of Infancy and Childhood*).



The *treatment* must aim at a betterment of the condition of the circulation through compensatory changes in the heart, and protection against external influences and physical over-exertion. Acute affections of the respiratory tract are especially to be feared. Attacks of cyanosis or threatened cardiac failure and dyspnœa will call for stimulation with either aromatic spirits of ammonia or brandy.

On general lines *Aconite*, *Arsenicum*, *Camphor*, *Cuprum*, *Digitalis*, *Glonoïn*, *Lachesis*, *Rhus tox.* and *Veratrum viride* are to be considered, their symptomatology covering the conditions met with in these cases, namely, hypertrophy, dyspnœa, excessive heart-action, cyanosis, etc. When symptoms are urgent spirits of ammonia will prove helpful.

#### PERICARDITIS.

Pericarditis in infancy is almost invariably seen as a complication of pneumonia, especially those severe pneumonias in which the pleura is notably involved (pleuro-pneumonia). Later on it will be seen secondary to rheumatism, pleurisy, scarlet fever and tuberculosis. Of all the causes capable of exciting pericarditis, rheumatism is the most important, and a certain amount of pericardial involvement is always to be suspected in severe cases of rheumatic endocarditis, although under these circumstances effusion seldom takes place.

Traumatism and caries of the ribs or vertebræ are local causes which may excite a pericarditis.

The effusion shows a strong tendency to become purulent, as do all effusions into serous membranes during childhood. The cases I have seen at autopsy, complicating pneumonia, were serous. Abundant fibrinous exudate is, as a rule, thrown out and a gluing together of the layers with complete obliteration of the pericardial sac is the usual unfortunate result.

The *pathological changes* noted elsewhere in inflammations of serous membranes are to be seen in pericarditis. The tendency to the pouring out of effusion, containing cellular elements in abundance, is pronounced. The dry stage is of



short duration. When the amount of fibrin, which covers the serous surfaces, is considerable and the effusion not sufficient to separate the layers, the heart presents a shaggy, irregular surface. The opposing surfaces may become adherent with a net-work of villous bands. As these bands of fibrinous exudate are absorbed they are replaced by granulation tissue rich in fibrinoblasts and permanent connective tissue formation results. More or less mediastinitis, as a rule, accompanies pericarditis in children.

Adhesions form to a greater or less degree in all cases which recover from the acute symptoms. This leads to a hypertrophy of the heart, or dilatation from interference with the nutrition of the myocardium. When absorption of the effusion is delayed, *myocarditis* develops, usually, leading to dilatation.

**Symptomatology.**—The early symptoms of pericarditis are rarely recognized in an infant owing to their obscurity and overweighing symptoms of the disease to which it is secondary.

If the child is old enough to complain of pain in the region of the heart, which may also be referred to as radiating to the left shoulder or epigastrium, or as occurring alone in these locations, a careful physical examination will reveal local tenderness and possibly cardiac friction-sounds, beside directing our attention to the fixation of the left side of the thorax. If friction-sounds are elicited, they will be heard as a rubbing or crackling sound synchronous with the heart's action and independent of respiration. They are most distinct under the fourth rib to the left of the sternum, and may simulate a mitral regurgitation murmur. However, cardiac friction-sounds do not only accompany the heart-sounds, but they are prolonged beyond them, being interposed and at times occupying the whole duration of the cardiac action (SKODA). In several cases I have heard the friction-sound most distinctly toward the base of the heart. Here it will persist even when moderate effusion has occurred, because this gravitates to the bottom of the pericardial sac.

With the appearance of the effusion the pulse, which was at first full and irritated, becomes feeble and irregular. Oppression, dyspnoea and cyanosis develop with the outpouring of sufficient fluid to embarrass the heart's action ; and eventually convulsions, and in older children delirium and coma, close the scene in fatal cases. A rapid outpouring of serum into the pericardium may produce sudden death. We sometimes see this occur during an attack of rheumatic fever and in pneumonia.

The pulse is of the greatest importance in recognizing acute inflammatory affections of the heart, being strongly suggestive of such a complication when irregularity and enfeeblement suddenly develop during an acute illness. The pulsus paradoxus may be present, but is not pathognomonic, as it may occur under other conditions in childhood (STEFFEN). Bulging of the præcordial region, increased area of cardiac dulness, and muffling of the heart-sounds and impulse are only to be elicited in severe cases. The two last signs are notably difficult to determine on account of the natural resiliency of the child's thorax and the greater accommodation possible under reverse conditions. The area of dulness is not triangular as in adults, and the heart, with its distended sack, retains its normal position, simply enlarging. Enlargement is more pronounced to the left. Unless dulness reaches up to the second interspace on the right side, it is more likely due to dilatation of the right ventricle than to fluid (KOPLIK). The percussion note is flat and resistant.

Adhesions are to be suspected when there is a displacement of the apex not due to marked hypertrophy, or cardiac dilatation and retraction of the intercostal space during systole. The mere retraction of the apex region during systole is by no means diagnostic of pericardial adhesions. When, however, associated with retraction of a considerable area of the thorax during systole, which rapidly returns to normal during diastole, we have strong evidence of the same (GERHARDT, *Lehrbuch der Auskultation u. Percussion*). Perhaps the most

conclusive sign is that pointed out by Broadbent, namely, retraction of the lower intercostal spaces posteriorly, due to tugging on the adherent diaphragm. The sudden rebound after systole produces a diastolic shock which is also pathognomonic taken in conjunction with the above signs. This is followed by a sudden collapse of the veins of the neck (FRIEDREICH). When thickening and contraction of the mediastinal structures, especially in the area surrounding the upper portion of the pericardium, is associated, there may be lessening of the calibre of the radial pulses and swelling of the neck veins during inspiration (KUSSMAUL). In many cases the diagnosis can be made as nearly as any physical signs permit by the following brief observations insisted on by Paul (*Diseases of the Heart*)—cardiac hypertrophy ; violent impulse of the heart, as a whole, but a feeble impulse of the apex. To this should be added, diastolic shock.

The *prognosis* of pericarditis is always grave, particularly when complicating pneumonia and scarlet fever. The likelihood of adherent pericardium resulting, which eventually produces myocarditis and dilatation, must be borne in mind.

**Treatment.** The child should be kept as quiet as possible during the active symptoms, and in case of recovery any physical exertion must be forbidden until every danger from cardiac dilatation is past. The ice-bag applied to the præcordium is of decided advantage in older children. Purulent collections in the pericardium which fail to become absorbed rapidly are less favorably treated surgically than pleural effusions, for which reason every effort should be made to overcome this condition remedially before resorting to aspiration.

**Acon.**—Chilliness ; hard, bounding pulse ; sharp pain in region of heart ; great restlessness and sighing ; dyspnœa and syncope. Useful in the earliest stages to control the vascular excitement.

**Arsen.**—Great anguish and oppression ; constantly changing position ; cyanosis ; thirst ; in consequence of repelled exanthems, or in connection with pneumonia ; stage of effusion

*Bryonia*.—This remedy follows well after *Aconite*, and is most applicable during the stage of effusion, although it seldom absorbs the exudate completely. *Sulphur* is a most valuable remedy for this purpose, especially when the case becomes protracted.

*Cactus grand.*—Sensation of constriction about the heart, as if a strong hand were grasping it. There may also be a sense of deep-seated soreness in the præcordium, with dyspnoea; attacks of suffocation; fainting; small, irregular pulse.

*Digitalis*.—Copious serous effusion; small, intermitting pulse; diarrhoea and vomiting; syncope.

*Iodium*.—Complicating croupous pneumonia. Violent palpitation and oppression from slightest motion; must lie perfectly quiet on back.

*Spigelia*.—After *Aconite*, when the friction-sound becomes audible. Sharp, stitching pains in chest. *Spigelia* is a most efficient remedy for the painful stage.

Besides these are to be considered *Asclepias tuberosa*, *Bell.*, *Cannab.*, *Canth.*, *Kali carb.*, *Lach.*, *Merc. cor.*, *Veratr. vir.*

#### ENDOCARDITIS.

Endocarditis is more liable to develop during the course of a rheumatic fever in children than in adults, but as the rheumatic condition is not as typical in children as in adults, this relationship is often overlooked. Likewise endocarditis is frequently associated with chorea and erythema nodosum, and recurring crops of subcutaneous fibrous nodules about the joints are taken as an indication of a progressive cardiac affection. Packard (*Amer. Jour. Med. Sci.*, Jan., 1900) notes five cases of acute tonsillitis and pharyngitis having no connection with rheumatism or any of its manifestations, in which endocarditis developed. While in these cases toxins absorbed from the throat might have set up structural changes in the endocardium by coagulation-necrosis or other chemico-vital action, still he inclines to the belief that the endocardium is directly infected with micro-organisms.

they gaining entrance by way of the tonsils. In support of this view is Charrins' case in which the staphylococcus aureus was found, both in the tonsils and in the endocardial vegetations.

Endocarditis is, therefore, in all probability, of infectious origin, being the result of infection with pyogenic cocci or with the pneumococcus of Fränkel. The tubercle bacillus may also set up acute endocarditis (VON RUCK). In simple endocarditis Säger and Fränkel have been able to demonstrate bacteria, and, according to Eichhorst (*Specielle Pathologie u. Therapie*), no distinction can be made between the group of micro-organisms capable of exciting the ulcerative variety in one case and simple endocarditis in another. The frequency with which the endocardium is attacked during the course of a rheumatic fever is readily interpreted by accepting in acute rheumatism an infectious disease, the result of specific bacteria, which attack with preference the serous membranes. Meyer (*Zeitschr. f. Klin. Medicin.*, Band lxxv., p. 311) found verrucose endocarditis present in twenty-one of one hundred animals injected with his diplostreptococcus. This micro-organism is probably identical with the one Poynton and Payne claim to be the cause of rheumatic fever.

Outside of rheumatism, endocarditis is seen with scarlatina, pneumonia, diphtheria, nephritis and septicæmia, in which case it is usually of a severe type. The ulcerative variety is so frequently associated with wound infection, and always partakes of the nature of a septic condition.

In simple or verrucose endocarditis the valves become covered with inflammatory excrescences—endocardial vegetations. Owing to destructive changes in the endothelial cells and consequent roughing of the surface of the valves these fibrinous formations are deposited from the circulating blood. At the same time the valves become thickened and distorted from interstitial cellular proliferation and vascular engorgement. Portions of the fibrinous vegetations may become detached and be swept into the general circulation, producing an em-

bolus at some distant point. The mitral valve is the most frequent seat of the endocarditic process, next in frequency being the aortic valves. Right-sided endocarditis has been discussed under *Fatal Endocarditis*.

In malignant, or ulcerative endocarditis the inflammatory state is more pronounced, being coupled with ulcerative and even suppurative processes in the endocardium.

**Symptoms.**—The onset of an endocarditis is always insidious, and especially when complicating another acute affection is its presence likely to be overlooked. Again, children rarely complain of pain or distress in the region of the heart, and a primary case may run its entire course unrecognized, being mistaken for some infectious disease, such as influenza or rheumatism. When associated with tonsillitis it is frequently overlooked. Subacute cases may run a long time with slight afternoon rise of temperature, progressive anæmia and loss of weight. Such cases are easily confused with tuberculosis and malaria. A routine examination of the heart in all febrile conditions is, therefore, imperative.

Endocarditis should be suspected if, during an acute infectious disease, there is an abrupt rise in the temperature with increased and weak pulse, præcordial distress and dyspnoea. In children of the rheumatic diathesis, a fever rapidly attaining a height of  $104^{\circ}$  to  $105^{\circ}$ , together with tonsillitis, is frequently accompanied by a severe endocarditis in the absence of all articular symptoms. The pulse, which at first is strong and possibly slow in comparison to the temperature range, soon becomes rapid and feeble, even dicrotic. A certain amount of myocarditis always accompanies endocarditis, and when cardiac weakness becomes extreme it should be suspected as a co-existing condition.

Præcordial distress and dyspnoea may be present, which, together with flushed face and the peculiarity of the pulse above referred to, are strong indications of this disease. The distress sometimes amounts to actual pain, which in young children may be referred to the epigastrium.



The pathognomonic symptom of endocarditis is the characteristic bruit, also described as the bellows murmur from its soft, blowing character. The murmur is systolic and is heard best at the apex. Endocarditis may, however, exist without this murmur being perceptible, as the subsequent course of the disease will show; and, again, during the infectious fevers a murmur is frequently heard, but it disappears during convalescence, leaving no trace of valvular defect behind, an

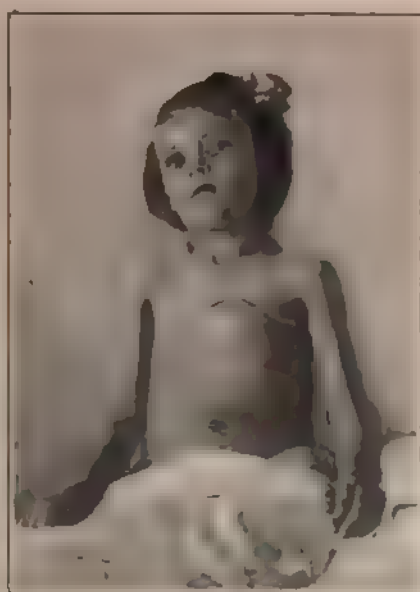


FIG. 43. ACUTE RHEUMATIC ENDOCARDITIS WITH DILATATION  
CYANOSIS AND DYSPNŒA MARKED.

autopsy entirely failing in these cases to demonstrate an inflammatory condition. If we study the murmur from day to day, we find that it gradually increases in loudness and distinctness, the first indication of its advent being a prolongation and blurring of the first sound of the heart.

According to Dr. O. Sturges (*Ashby and Wright*), a faint murmur heard at the top of the ensiform cartilage, indicating



regurgitation at the tricuspid orifice, due to back pressure through the lungs, can in some cases be heard to precede the mitral bruit.

With *malignant or ulcerative endocarditis* the symptoms of septicæmia become prominent; the temperature is intermittent, and there is enlargement of the spleen and albuminuria beside a strong tendency to embolus formation in the brain or in other important organs. Such cases are fatal, as a rule, while in a well-managed case of simple endocarditis the prognosis as to life is always favorable, but the ultimate outcome as regards permanent valvular defects is a question. There is no doubt that in some cases, under proper treatment, the murmur will entirely disappear and there will be no evidence of valvular leakage or obstruction later on. On the other hand, endocarditis may produce such general damage to the endocardium as to blight the child's existence permanently and lead to early death. A comparatively slight lesion, also, by producing mitral stenosis, will do much more harm than one simply causing a leak at this valve. Another important point to be remembered is the strong tendency to recurrence in endocarditis, especially in rheumatic subjects. Every new attack adds to the existing damage.

**Treatment.**—An essential element in the successful treatment of endocarditis is absolute rest, as any physical exertion capable of exciting the heart to increased action will necessarily exert a baneful influence upon the inflammatory process. The body surface must be carefully protected against chilling influences, and long-continued rest, even during convalescence, is at times imperative, particularly when myocarditis is suspected.

The ice-bag applied for fifteen minutes to half an hour, every two hours, is a valuable adjuvant during the acute stage.

Prophylaxis is of importance. Children subject to rheumatism should be kept under constant vigilance, and their diet and dress carefully regulated. Enlarged tonsils and adenoids should be removed.

*Aconite*, *Belladonna* and *Veratrum viride* in the early stages, and later *Spigelia*, *Spongia*, *Cactus*, *Bryonia* and *Colchicum*, are the most important remedies. After the acute symptoms have subsided much of the damage to the heart naturally to be expected can be prevented and overcome by the judicious choice of a remedy capable of absorbing the inflammatory products and correcting the resulting disturbances. It is not difficult to obtain sufficient data for such a prescription, and here *Aurum*, *Iodium*, *Spongia*, *Sulphur*, *Calc. carb.*, *Lachesis*, *Arsenicum* and *Arsenicum iod.* are the most frequently indicated drugs. *Kali mur.* exerts a specific action upon the heart-muscle, and is recommended to avert dilatation (ARNULPHY).

*Acon.*—Chilliness; hard, wiry pulse with high fever, restlessness and dyspnœa. *Veratrum viride* has less of the restlessness; the arterial tension is extreme and cerebral symptoms may supervene, and, although it controls excessive cardiac action promptly, its influence upon the inflammatory process and the fever is inferior to that of *Aconite*.

*Belladonna.*—Full, bounding pulse, flushed face, skin hot and moist, delirium.

*Bryonia.*—Purely rheumatic cases; pericarditis and endocarditis; sharp pains at heart, relieved by lying upon the affected side; tongue dry and coated; great thirst; no desire to move.

*Cactus.*—Sense of constriction in region of heart; oppression of breathing. (See *Pericarditis*.)

*Colchicum.*—Rheumatic endocarditis; tearing pain in region of heart; small, thready pulse (JOUSSET).

*Iodium.*—Purring sensation in region of heart on palpation; violent palpitation and dyspnœa, even to fainting, on slightest exertion, with pneumonia; if *Spigelia* does not give relief within a reasonable period of time (KAFKA).

*Kali carb.*—Blowing systolic murmur with accentuated pulmonary sound; pulmonary engorgement; weakness of the heart-muscle, with anasarca of feet and ankles; associated myocarditis; after *Bryonia*.

*Spigelia*.—Considered by some the most important remedy in endocarditis. It may be given as soon as the condition becomes recognizable, in the absence of strong indications for another remedy. Personally I prefer *Bryonia*, as I consider it more closely related to the pathologic process. There is no doubt, however, that *Spigelia* is a most valuable remedy in many cases of acute heart pain.

*Spongia*.—Paroxysms of oppression and pain in the heart ; inability to lie with the head low, or even complete inability to lie down on account of the choking paroxysms induced thereby.

#### MYOCARDITIS.

Acute degenerative and inflammatory changes in the heart-muscle are of frequent occurrence in the acute infections of childhood. The toxins of diphtheria, scarlet fever and typhoid fever are especially concerned in the production of myocardial degeneration (ROMBERG). True inflammatory changes—myocarditis—are most frequently associated with endo- and pericarditis, and are due to the invasion of the heart-wall with pyogenic organisms, chiefly the streptococcus pyogenes, staphylococci and the pneumococcus (ZIEGLER). Myocardial changes have also been observed in whooping cough by Koplik and Osler. Pyrexia is a contributing cause, but does not seem able to produce myocarditis by itself.

The varieties of degeneration encountered are granular, hyaline and vacuolar. All of them may have more or less fatty changes associated. The process may be purely degenerative throughout, but, as a rule, exudation and cell proliferation in the connective tissue stroma is associated therewith. In infectious and pyæmic cases areas of round cell infiltration play a prominent rôle, which may break down, resulting in small intramural abscesses.

At autopsy the heart is found of a pale, yellowish-brown, turbid color and the muscle is easily torn. It is the soft-heart of the older writers. The process is mostly diffuse, although—

in true myocarditis the changes may be more pronounced in different areas.

The *symptoms* are essentially those of a weak heart. When myocarditis develops during the course of typhoid fever or pneumonia we realize that the pulse is too thin and rapid, the disproportion in the respiratory ratio too pronounced and the first sound too weak to be accounted for merely by the fever. In the absence of demonstrable peri- and endocarditis we feel that here we have to deal with a degenerated myocardium. In the course of diphtheria the child is suddenly seized with epigastric pain, vomiting, syncope; rapid, irregular pulse. A much worse prognosis than such a condition cannot well be named.

The softening of the heart-muscle invites dilatation; there is, therefore, usually some dilatation, especially of the right ventricle. A faint apical systolic murmur may be present. The heart is usually rapid and embryocardiac in rhythm. Bradycardia may develop, especially after diphtheria. On the other hand, there may be no symptoms, or only a short time before death will there be sufficient indications to make us suspect myocarditis.

Chronic myocarditis presents the symptoms described under Chronic Heart Disease as "failing compensation."

The *diagnosis* of myocarditis cannot always be made during life, but there are certain symptoms that strongly point to its existence. The subject is well summarized by Koplik (*Med. News*, March, 1900) as follows: Attacks of faintness, pallor, vomiting; disturbed and irregular heart's action; persistent distortion of the respiration and pulse-ratio as in adherent pericardium. When these attacks show a tendency to recur they are certainly significant. Physical examination reveals a weak apex-beat, weakness of the first sound or loss of its muscular quality, greater intensity of the second sound at the apex and accentuation of the pulmonary second sound. In pertussis there is in addition slight systolic blow at the apex, oedema of the face and extremities, pallor, cyanosis and drowsiness.

The *prognosis* is grave. Under long-continued rest the heart may regenerate sufficiently to resume its function as before, providing the changes have not been too extensive. The symptoms described as indicative of myocarditis are in reality due to dilatation (OSLER). The abrupt death in the course of an acute infectious disease results from cardiac paralysis.

The *treatment* calls for the most complete rest. As long as symptoms show the slightest tendency to recur the child should not be permitted to feed itself or make the slightest physical exertion. The remedy most homœopathic to the degenerative changes is *Phosphorus*, and it is no doubt of value. I have certainly seen it benefit cases of this class. Alcohol should at the same time be given in moderate amounts. As an emergency remedy, Holt speaks highly of *Morphia*.

#### CHRONIC VALVULAR DISEASE.

Chronic acquired valvular disease is the sequel to inflammatory affections of this organ, notably acute endocarditis. The lesions which may be encountered are: (a) *Thickening and distortion of the valves*; (b) *Fibrinous or calcareous deposits upon the valve-leaflets*; (c) *Hypertrophy of the walls*; (d) *Dilatation of the chambers*; (e) *Adherent pericardium*. These changes are usually seen in various stages of development, and in pronounced cases they may all be demonstrated in different portions of the organ. The mitral valve is by far the most frequently affected seat of lesion, the aortic valve being rarely affected in children, and, when so, more often in association with mitral disease than alone. The changes in the valves above referred to lead either to regurgitation or obstruction at the orifices. Both conditions may exist at one orifice, so that it is not uncommon to find mitral stenosis and regurgitation in the same patient.

**Symptoms.**—The history of an organic heart affection can be described in three stages, constituting the classical course

pursued by this disease. The *first stage* marks the onset, being the acute inflammatory stage, which leads either to immediate damage to the valves or to chronic endocarditis. The rheumatic diathesis underlies the vast majority of all cases of recurring or chronic endocarditis, and it is usually possible to obtain a previous history of rheumatic symptoms, such as recurring acute tonsillitis with joint pains, arthritis, erythema, fibrous nodules, chorea, etc., or to note the later development of one of these conditions in a case of valvular heart disease.

The *second stage* is that of compensation, during which the heart adapts itself to the extra strain brought upon its muscular walls incident to the leakage or obstruction at its orifices. This is accomplished through hypertrophy of the ventricular walls, and a compensating heart is, therefore, usually an enlarged or hypertrophied organ. When compensation is perfect there are naturally no symptoms; but as this is not always the case, the patient suffering more or less from shortness of breath on exertion, palpitation, attacks of epistaxis, bronchitis, indigestion.

*Third stage.*—The stage of failing compensation is the period at which the heart becomes incompetent to maintain the circulation, in consequence of which the arteries are but imperfectly filled with blood and the veins become engorged. Although the patient may be abruptly thrown into this stage by undue physical exertion or a fresh attack of endocarditis, pneumonia, typhoid fever or scarlet fever, still the usual course is that of progressively-increasing cardiac weakness, hastened by impairment of the general nutrition, anæmia, intercurrent diseases, etc.

The symptoms of cardiac incompetency, when of gradual onset, will show themselves in dropsy of the lower extremities; difficult breathing from the slightest physical exertion and when lying with the head low; cough, with frothy, blood-streaked expectoration; flatulent indigestion; scanty, albuminous urine of high specific origin. When of sudden onset



there is marked dyspnœa and cyanosis; the lungs are the seat of venous engorgement, which frequently leads to pulmonary œdema and death.

The imperfect circulation resulting from valvular disease interferes with the general nutrition and sets up important visceral changes. To the former belong clubbing of the fingers and stunted growth, and to the latter, chronic bronchitis, chronic congestion of the spleen, liver and kidneys.

The *prognosis* of organic heart disease is never favorable, as complete recovery is impossible, and the possibility of renewed attacks of endocarditis and other factors capable of rupturing compensation must be a constant menace to the child's condition. The course is usually a progressive one, and puberty seems to exert an unfavorable influence upon the disease. Nevertheless, well-managed cases may attain adult life with safety, and by the maintenance of a good general nutrition develop no serious symptoms.

#### MITRAL REGURGITATION.

Mitral regurgitation is the commonest valvular defect of childhood, resulting from distortion, and consequent imperfect closure, of the mitral valve. Owing to the regurgitation of the blood into the left auricle, the same becomes hypertrophied, and later dilated; the pulmonary circulation becomes embarrassed and an extra amount of work is thrown upon the right ventricle, which also hypertrophies in order to meet the extra strain upon its walls. The damming back of the blood in the pulmonary artery causes the *accentuated second sound* over the pulmonary valve, so characteristic of mitral regurgitation. The left ventricle eventually hypertrophies, in consequence of the increased pressure in the pulmonary artery, against which it must work in order to sustain the circulation. Urgent symptoms are the result of failing right heart, the right ventricle often dilating to a great degree, even to the production of incompetency of the tricuspid valve.

The *physical signs* of mitral regurgitation are a systolic



murmur heard with the greatest intensity at the apex and transmitted into the left axilla; accentuated second sound over the pulmonary artery; increased area of dulness to the right, indicating hypertrophy and dilatation of the right ventricle; displacement downward and outward of the apex.

#### MITRAL STENOSIS.

Mitral stenosis is frequently associated with regurgitation, owing to a shrinkage of the valves and the auriculo-ventricular orifice, or obstruction resulting from fibrinous or calcareous deposits. In quite a number of instances, however, it exists alone, and in such cases it is generally associated with sub-acute rheumatism and insidious endocarditis. There are cases in which we can get absolutely no history of rheumatism or any other preceding infectious disease.

A typical case of mitral stenosis is marked by dyspnoea; small, feeble pulse; dilatation of the left auricle, and hypertrophy, with later dilatation of the right ventricle, the left ventricle not participating in the process unless regurgitation is associated. The physical signs are a presystolic murmur, which may assume a purring character, perceptible to the touch; sharp, snappy first sound at the apex; accentuated second sound over the pulmonary valve; area of dulness increased upward and to the right. The presystolic thrill is the most characteristic of all signs in valvular heart disease, and upon this symptom alone—if we can exclude adherent pericardium—the diagnosis is readily made. It is best felt by placing the flat hand over the cardiac area, the thrill being plainest in the fourth interspace to the left of the sternum.

Mitral stenosis is more frequently found in phthisical subjects than the other forms of valvular disease. Indeed, mitral regurgitation is looked upon as unfavorable to the development of phthisis.

## AORTIC STENOSIS.

Stenosis of the aortic orifice results from pronounced attacks of endocarditis, for which reason it is one of the rarer organic affections and seldom seen alone, usually being associated with mitral regurgitation. From the nature of the lesions at the aortic orifice, regurgitation is also frequently added to the obstruction. Unless there is marked stenosis, symptoms are not prominent, as the hypertrophied left ventricle perfectly compensates for the defect. Complete recovery is possible.

The physical signs are a systolic murmur heard over the aortic orifice and transmitted into the carotids; displacement of the apex downward and outward from hypertrophy of the left ventricle; slowing of the pulse.

## AORTIC REGURGITATION.

Regurgitation of the aortic orifice, like stenosis, is rare in children, and is never observed as a single condition. The commonest causes for aortic regurgitation, namely, sclerosis of the valves due to syphilis, gout and alcoholism, are practically never present in children, and for this reason it is only found with severe cases of endocarditis, especially the variety complicating the infectious fevers, from which stenosis and mitral disease also result. In a case which came under my notice the valve was found unaffected by inflammatory deposits at the autopsy. There was, however, mitral disease and dilatation. The insufficiency at the aorta was, therefore, most likely relative, *i. e.*, due to dilatation of the aortic orifice.

As a result of the regurgitation of the blood into the left ventricle the same becomes markedly hypertrophied, later dilating with the consequent production of mitral regurgitation.

The physical signs are a rapid, strong, full pulse, with sudden collapse (Corrigan's pulse); a diastolic murmur at the aortic orifice; extension of cardiac dulness in the direction of

the heart's long axis and displacement of the apex-beat downward and outward; arched appearance in the region of the præcordium in young subjects; strong, bounding pulsation of the carotids. The capillary pulse can also be demonstrated at the matrix of the finger-nail or by pressing a glass slide against the forehead. Riesman has on several occasions noted pulsation of the uvula. The water-hammer pulse is best observed by grasping the forearm with both hands just above the wrist and holding it in a vertical position. The rebound, or diastolic shock, is then plainly felt.

**Treatment.**—The child's general condition must be carefully observed and physical overexertion strictly prohibited, in order to maintain as perfect a compensation as possible. Systematic exercise, with sufficient sleep and a highly nutritious, non-stimulating diet, and special attention to the digestive function, are the important considerations.

No form of treatment has as yet exceeded in expectations the results obtained by the Schott method of baths and resisted movements, and when given in conjunction with remedies which may be called for upon general or special indications, this line of treatment will, no doubt, prove itself without equal. The effect of the bath, as described by Dr. Edward R. Snader (*Hahnemannian Monthly*, November, 1898), is "to reduce the size of a dilated heart, diminish the number of pulse-beats, fill the arteries, partially empty the veins, open the cutaneous capillaries, and inaugurate a rehabilitation of a damaged heart-muscle by reason of nutritional change." That this change takes place in the heart has not only been repeatedly demonstrated by the skillful use of percussion, but it has also been actually shown by means of skiagraphs, personally made by Dr. Theodore Schott (*Medical Record*, March 26, 1898).

The advent of urgent symptoms as a result of ruptured compensation will at times require a purely physiological prescription to tide the case over. Pulmonary congestion due to overfilling of the right heart and general venous stagna-

tion calls for *Glonoïn* (drop-doses of the second decimal dilution, repeated half hourly until relief is obtained), the action of which is well supplemented by *Veratrum viride* 1x, given at less frequent intervals. A failing left ventricle is frequently rescued by the judicious use of *Digitalis*, in five-drop doses of the tincture, repeated in from three to four hours until results are obtained. Under no circumstances, however, should such treatment be extended beyond the critical period. Rest, tonic treatment and a carefully-chosen remedy must be relied upon to safely bring the patient back to a state of restored compensation. *Ferrum* in one of its forms when there is anæmia and *Cinchona* to improve the appetite and general condition are indispensable. Dr. Snader highly lauds *Cactus* as a safe, efficient heart tonic, which may be used without fear of producing overstimulation or cumulative effects. The *Iodide of Arsenic* is also a valuable heart remedy when there is shortness of breath on exertion and slight œdema of the ankles.

Attacks of sudden heart failure call for rapid-acting stimulants, such as *Ammonia*, the *Alcoholics*, and *Nitroglycerin*. The most positive results are obtained in these cases by the hypodermatic injection of *Strychnia* (one one-hundredth to one-fiftieth of a grain).

*Acon.*—Attacks of anxiety, pallor, restlessness, tingling in the extremities, small, thready pulse, fear of death. Also hacking cough, with stitching pains and hæmoptysis accompanying valvular disease.

*Arsenicum.*—Cardiac weakness with præcordial anguish and oppression; inability to lie down; nocturnal aggravation; anasarca of lower extremities. *Arsenicum iod.* embodies to a certain extent the properties of *Arsenic* and *Iodine*, both of which possess marked and characteristic cardiac symptoms in their pathogenesis.

*Cactus grand.*—*Cactus* is quite extensively used in the first decimal dilution, and in the tincture, for its sustaining action upon the heart, being credited with the non-production

of cumulative or harmful effects. It is a valuable remedy in mitral disease with pains radiating down the left arm ; also sense of constriction about the heart ; small, feeble, intermittent pulse ; icy-cold feet.

*Convallaria*.—Mitral stenosis, with dyspnœa and irregular heart action ; dilatation of the right ventricle.

*Digitalis*.—Irregular, intermittent action of heart. During perfect rest the heart's action is slow, but the slightest exertion produces accelerated and irregular action. Sensation of complete arrest of heart's action.

*Gelsemium* is similar in some respects to *Digitalis*. There is a feeling as if the heart would stop beating if he did not keep moving about ; also asynchronism of heart's action and cyanosis of the lips.

*Iodium*.—Violent palpitation from the slightest exertion. Shortness of breath, palpitation and feeling of weakness on going up-stairs ; constant heavy, oppressive pain in the region of the heart (HERING).

*Lachesis*.—Awaken from sleep with sense of suffocation in throat ; cannot bear anything tight about throat ; venous congestion of internal organs ; dilated veinules on chest ; defective peripheral circulation and tendency to cyanosis.

*Natrum mur.*—Fluttering of the heart, with attacks of faintness ; irregular and intermittent heart's action ; anæmia.

*Phosphorus*.—Tightness across the upper portion of the chest, with tight cough and spitting of blood ; weakness of the right heart, with venous stagnation ; cannot lie on the left side ; palpitation from every emotion, with rush of blood to the chest in rapidly developing children (*Calc. phos.*).

*Rhus tox.*—Rheumatic affections ; hypertrophy from physical overexertion, with palpitation and pain shooting from region of heart down the left arm.

*Spigelia*.—Sharp, stitching pains in region of heart ; anxiety and oppression when lying down ; can only sleep on the right side ; purring feeling over heart. Great dyspnœa at every change of position.

*Spongia*.—Attacks of oppression and cardiac pain when lying with head low ; suddenly awaking after midnight, with suffocation, great alarm, anxiety (HERING). Valvular insufficiency, pericarditis in stage of effusion and aneurism are within the scope of this remedy.

In the *dropsy* of heart disease I have obtained the best results from *Apocynum cannabinum*, twenty drops of the decoction (BOERICKE & TAFEL'S) three times daily. Swelling of the ankles and other minor conditions, simply pointing to a weak heart and sluggish venous circulation, yield very satisfactorily to such remedies as *Arsenicum*, *Bryonia*, *Kali carb.* and *Lycopodium*. Purgation may become necessary in pronounced cases, threatening the patient's life.

#### FUNCTIONAL DISORDERS.

As has already been pointed out, functional disorders of the heart are not very common during childhood. The most prominent causes of these disorders are gastro-intestinal irritation, helminthiasis, teething, anæmia, chorea and hysteria.

The *symptoms* are various, the commonest type of disorder being irregularity of rhythm and intermission; palpitation with rapid breathing and attacks of syncope being next in order. Heart consciousness is obviously less common in children than in adults, and angina pectoris is quite a rare disease.

A case of neurotic *angina pectoris* in a girl ten years old was brought to my clinic at the Children's Homœopathic Hospital. She was apparently in good health, having had no prior serious illness, and the family history was good, with the exception of rheumatism on the mother's side. There had been stitching pains in the heart for several weeks, and two weeks before she was seen the first paroxysm developed. The paroxysms then appeared at intervals of several days, beginning with a feeling of extreme tiredness and oppression about the heart, followed by sharp agonizing pains in the heart and pains radiating down both arms into the wrists, the

left one becoming affected first. The face became flushed, the pulse small and rapid, and the heart's action violent. *Amyl nitrite* inhalations immediately controlled the paroxysms, and as she had three in succession at the hospital the diagnosis could be readily confirmed. Under the administration of *Spigelia* there was no further return of the trouble.

The *treatment* of functional heart affections is mainly hygienic, coupled with the administration of such remedies as *Aconite*, *Belladonna*, *Cactus*, *Digitalis*, *Kalmia*, *Nux vomica*, *Pulsatilla*, *Rhus tox.* and *Spigelia*. The child's general condition must be looked after, and if helminthiasis, intestinal catarrh, lithæmia or any other exciting cause can be discovered, treatment should be instituted in that direction.



## CHAPTER XIII.

### DISEASES OF THE KIDNEYS AND URINARY TRACT.

Nephritis complicating the infectious fevers is the most frequent form of renal disease encountered during childhood. Malformations are occasionally encountered at autopsy but cannot be diagnosed. Innocent fibromata, adenomata and cystic degenerations are occasionally met with, and outside of hæmaturia produce no characteristic symptoms, unless they attract attention by attaining considerable size. Infants with cystic kidneys die shortly after birth as a rule. The condition is almost always bilateral.

Malignant tumors are most frequently sarcomata and have a tendency to grow very rapidly. Carcinoma of the kidney is also relatively common in children. It is estimated that 38 per cent. of all new growths of the kidney reported occurred in children (LEWIS). Hydronephrosis may occur during childhood; cases have been observed which evidently depended upon phimosis.

The kidney in the infant is about twice as large—compared with the body weight—as in the adult. In shape it is more lobulated. In the new-born, deposits of sodium urate—uric acid infarcts—are frequently encountered in the tubules on making a section of the organ. The kidneys can occasionally be palpated in children with flabby abdominal walls and lax tissues, especially in the rachitic, the right kidney being the most accessible. It is hardly fair to speak of such a condition as *floating kidney*, but a number of authentic cases of this nature have been recorded. Koplik speaks of a case in a girl eight years old in which there was present epigastric pain and hysterical manifestations.

The urine must be studied both from the chemical and microscopic standpoint and the frequency of urination. Total

daily quantity, the presence of pain, fever, loss of control over the bladder and association of other symptoms pointing to impaired renal function must also be taken into consideration in making a diagnosis in diseases of the urinary tract. During infancy there is a relatively larger quantity of urine excreted than later in life. Standard figures cannot be given, but the amount gradually increases from *one ounce* or more in the first week of life to about *twenty ounces* at the completion of dentition, and *thirty-six ounces* just prior to puberty.

The *frequency of urination* depends upon the age, and, to a less extent, upon such modifying influences as sleep, temperament and habit. Concerning the *control over micturition*, Ulzmann (*Genito-Urinary Neuroses*) writes: "In the earliest childhood urination and defecation take place without any subjective sensations. The slightest contraction of the bladder and of the rectum suffices to expel the urine and fæces, since the resistance of the sphincters is wanting. After the first year of life children begin to voluntarily hold back the fæces, while the urine still flows involuntarily, often against the will of the little ones. The ability to hold the urine back at will is usually established at about the end of the second year, that is, after the first dentition."

The *specific gravity* is lower than in adults, and the urine contains a greater percentage of uric acid, but less urea and inorganic salts. It is usually clear, but may be turbid from the presence of mucus and white urates.

The *method of collecting the urine* for examination has been described in the chapter on "Clinical Examination."

#### ALBUMINURIA; CYCLIC ALBUMINURIA.

Albuminuria not traceable to nephritis is encountered in two forms, namely, *idiopathic albuminuria* and *acute degeneration of the kidneys*. The former condition is also known as *cyclic* or *orthostatic albuminuria*. The latter name well expresses the nature of this form of albuminuria, which disappears on complete rest and reappears after the patient has

been up and about for some time. Physical exertion increases the amount of albumin. It is most frequently seen in male children who are anæmic or neurotic, and develops during adolescence. Heubner looks upon cyclic albuminuria as a condition without danger that ultimately disappears, while Senator thinks that it always indicates some insidious structural changes in the renal parenchyma. It is difficult to say in the beginning just how a case will terminate.

*Acute degeneration of the kidneys* occurs during the infectious fevers, especially in scarlet fever, diphtheria, pneumonia and typhoid fever. Henoch has also repeatedly seen this condition in autopsies upon atrophic children, children dying from diseases characterized by marked loss of vital fluids, *e. g.*, cholera infantum, intestinal tuberculosis, etc., and after prolonged high temperature. Morse (*American Med.*, April 5, 1902) found acute degenerative nephritis in 15 per cent. of a series of seventy cases of enteric diseases in infants. No symptoms outside of the urinary findings were present to suggest the condition. Personally, I have not seen decided structural kidney changes in autopsies upon infants dying of enteritis as frequently as some writers report.

Certain poisons, like *Arsenic* and *Phosphorus*, and many drugs, notably *Cantharis* and *Turpentine*, act upon the renal epithelium while being eliminated. The local as well as the internal use of *Bichloride of Mercury* is frequently attended with albuminuria. It is questionable whether there be such a condition as *febrile albuminuria*, where the action of toxins can be excluded.

The kidney appears slightly swollen, and the cortical substance presents a grayish appearance, which may advance to fatty changes. Microscopically the epithelial cells of the tubules are the seat of cloudy swelling.

The *prognosis* is favorable in both conditions. In idiopathic albuminuria much can be done by constitutional treatment. The diet is of great importance, and frequently a restriction to farinaceous foods, fish, fruit and fats suffices to

clear up the albuminuria, as demonstrated by Fothergill (*Manual of Dietetics*). Where, however, the patient is poorly nourished, a tonic treatment must be instituted, together with the constitutional remedy. Febrile albuminuria clears up with the recovery from the disease which has induced it. Nitrogenous food must be withheld during its course, and in the absence of strong indications for another remedy, one of the following should be selected:

*Apis, Arsen., Canth., Merc. cor., Terebinth.* For special indications, see Treatment of *Acute Nephritis*.

#### ŒDEMA WITHOUT KIDNEY LESION.

In protracted cases of enteric disturbances it is not uncommon to find more or less general œdema without the slightest trace of nephritis. There is puffiness of the eyelids and a cushion-like swelling on the dorsum of the hands and feet. It may even involve the extremities. This is associated with anæmia and feeble circulation. It is undoubtedly only a symptom of weakness. Changes in the lymphatic system resulting from the absorption of toxins are held to exist by some writers.

In all conditions of anæmia, notably in chlorosis and hydræmia, there is a tendency to anasarca. In the terminal stage of tuberculosis we often see it.

#### HÆMATURIA; HÆMOGLOBINURIA.

*Hæmaturia*, or blood in the urine, has the same significance in infancy as in later life, although it is much less frequently due to organic and mechanical causes (papilloma, calculus) than to acute nephritis, tuberculous cystitis and general disturbances, such as hæmorrhagic disease of the new-born, purpura, scurvy. Of the last named condition it may be the first and most prominent symptom; in fact, the only symptom.

*Hæmoglobinuria*, or hæmoglobin in the urine, results from the action of some toxic agent or ferment upon the blood, through which the hæmoglobin is dissolved out of the corpuscles and excreted with the urine.

It has been observed in various infections (malaria, scarlet fever), in helminthiasis, after exposure to cold and as a result of certain drugs (*Potassium chlorate*, *Phosphorus*, *Arsenic*). The most striking form is *recurring hæmoglobinuria*. This usually affects children whose health is below par, and in many cases there is a history of hereditary syphilis. I have had such a case under observation for a long time. This child also had scarlet fever. The attacks occur at irregular intervals and are associated with anorexia, malaise and other general disturbances. The urine is of a dark reddish-brown color, high specific gravity and contains albumin. The disease is usually outgrown.

The *treatment* is symptomatic.

#### ACUTE NEPHRITIS.

Pathologically, two forms of acute nephritis may be distinguished, based upon the intensity and location of the inflammatory process. In the milder form there is congestion of the kidneys, with exudation of blood plasma and leucocytes, and degeneration of the epithelium of the urinary tubules and glomeruli. For this variety Delafield has chosen the name *acute exudative nephritis*, while the more extensive inflammation, in which the stroma and glomeruli are involved in permanent pathological changes, he designates as *acute productive nephritis*. Here there is, beside the exudation, growth of the capsule cells of the Malpighian bodies. This form of nephritis is especially common in older children, occurring as a complication of diphtheria and scarlet fever (COUNCILMAN).

Macroscopically the kidney may show but slight evidence of congestion or degenerative changes. Usually it is swollen; softer and more moist than normal. The cut surface is grayish or mottled red, indicating degenerative changes and alterations in the distribution of the blood. The cortex is light or yellowish-gray, while the medullary portion (pyramids) is cyanotic. The glomeruli may be seen as red or grayish points.

**Etiology.**—The relation of the nephritis of childhood to infectious fevers is almost an inseparable link ; indeed, cases not traceable to one of these fevers are looked upon as the result of an infection of unknown origin. A few cases of *primary nephritis* are on record. The most important contribution on this subject is from Holt (*Archives of Pediatrics*, 1887). In this form of nephritis the renal symptoms may be insignificant as compared with other associated symptoms such as enteric disturbances, for which reason it is no doubt frequently overlooked.

The pathological changes taking place in the kidney parenchyma vary with both the virulence and the amount of toxin circulating in the blood and are as follows : *Acute degeneration* ; *acute exudative nephritis* ; *acute productive nephritis*.

The first-named condition is the primary result of the toxins, and, if the irritation is not prolonged beyond a certain point, it will be the only pathological change taking place. Acute degeneration, therefore, appears in the early stages of infectious fevers.

Should, however, the irritation be prolonged, or the exciting cause be quite energetic in action from the beginning, a true exudate inflammation will be the result. For this reason actual nephritis is usually a later occurrence in the course of the fever.

The productive type of nephritis is a more diffuse inflammation, more subacute in its type, and most frequently follows upon *scarlet fever*, as a result of the powerful kidney-poison peculiar to this disease. Pneumonia is more likely to produce nephritis in childhood than in adult life. The same may be said of influenza.

Aside from the clearly infectious cases, nephritis has been attributed to catching cold from exposure to draughts or living in cold, damp dwellings ; the presence of bile-acids in the blood ; diphtheria antitoxin injections, and to the use of many well-known drugs.

**Symptomatology.**—Occurring early in the course of a severe infectious fever, the presence of albumin in the urine, together with a few hyaline or granular casts, indicates nothing beyond an acute degeneration of the kidneys. This may also occur during prolonged high fever; but when a true nephritis develops there are added the symptoms of dropsy, scanty urine, increased fever, and the presence of renal epithelium, blood, leucocytes, hyaline and granular casts.

A primary nephritis is ushered in with high fever, pain in the region of the kidneys, headache and vomiting, scanty urine and dropsy.

When secondary to an infectious fever the symptoms develop less abruptly. They make their appearance at the height of the fever or during convalescence as sometimes takes place in scarlatina. Frequently a renal affection is not suspected until dropsy and scanty urine become prominent, or until the protracted course of the disease leads to an examination of the urine, when the mystery becomes solved. Post-scarlatinal nephritis appears, as a rule, in the third or fourth week of the disease.

Dropsy is naturally most noticeable in those portions of the body possessed of loose areolar tissue, and for this reason the face, particularly the eyelids, the wrists and ankles, legs and scrotum, become most markedly affected. The pleural and peritoneal sacs are involved in grave cases. (See Fig. 44.)

Dilatation of the heart, indicated by an increase in the area of cardiac dulness and weak pulse, is a frequent complication arising during the course of nephritis. The urine is diminished in quantity, the specific gravity high, although the amount of solids excreted is far below the normal. Its color is dark-red or smoky, the latter indicating the admixture of renal blood, and it contains albumin in abundance; blood; leucocytes; renal epithelium and casts. Early there will be blood and narrow hyaline casts; later epithelial, granular and broader hyaline casts.



**Prognosis.**—The absence of complete suppression of urine and uræmia, (the latter condition manifesting itself as severe headache and vomiting, followed by coma and general convulsions,) and the speedy control of the anasarca, with absence of extensive pleural effusions and œdema of the glottis, offers a favorable prognosis as far as the acute condition is concerned.

Long-continued anæmia and albuminuria indicate interstitial and destructive changes in the kidney structure, but in view of the fact that the child's kidney possesses marked regenerative power, recovery can often be expected under extreme care in hygienic measures and in the selection of

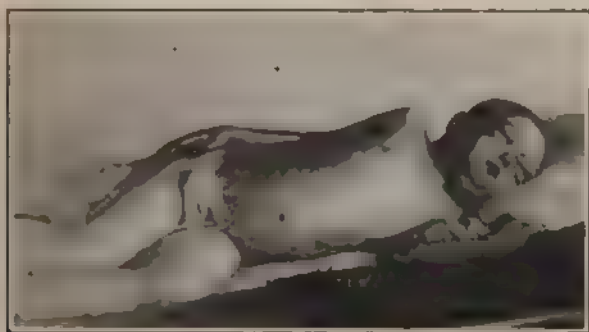


FIG. 44 —A CASE OF ACUTE (POST-SCARLATINAL) NEPHRITIS WITH ANASARCA AND ASCITES.

remedies. It is, indeed, remarkable to witness the evidently complete recovery that so frequently takes place in severe nephritis.

Our therapeutic resources are particularly rich in the symptomatology of renal affections, both for the acute as well as more permanent changes taking place in the kidney, and the results of careful prescribing are most gratifying.

**Treatment.**—As most of the cases develop during one of the infectious fevers, prophylactic measures stand high in the therapy of nephritis. These must aim to spare the kidneys as much as possible from any extra amount of work and from

the danger attending congestion of these organs, the practice of which resolves itself into the elimination of highly nitrified foods from the dietary ; a maintenance of the normal spontaneous function, or even a stimulation of the same ; rest in bed and protection against chilling influences. Especially in scarlet fever is it imperative to keep the child in bed for from three to six weeks, according to the severity of the case, and return most cautiously to a diet of solid food.

The free use of boiled water and the daily warm bath to induce gentle sweating under a woollen blanket is a great aid to the kidneys, and frequently all-sufficient to overcome moderate anasarca. Suppression of urine and uræmic manifestations call for the hot pack. In the presence of anasarca and suppression of urine the drinking of water must be temporarily cut down to a minimum. Goodno recommends the hot-air bath in these cases.

Personally, I place the greatest reliance upon hot high rectal enemata in conjunction with the hot pack in acute nephritis with suppressed or scanty urine and uræmic symptoms (vomiting ; cerebral irritation). These injections act as a stimulant to the abdominal sympathetic and by inducing free diuresis relieve the renal congestion and eliminate toxins. The diet should consist mainly of milk, and in no case should meat, eggs or strong broths be administered. Cereals and stewed fruit may be selected as solid food is gradually resumed.

Owing to the fact that the nephritis is usually secondary to some other condition, our indications for a remedy are not so sharply defined as in the primary form of adults.

*Arsenicum* is indicated by the great anæmia and anasarca especially prominent about the eyelids in the morning. There is scanty urine, the characteristic thirst and restlessness, and cardiac involvement.

*Apis* is frequently called for, and is most useful for conditions which arise suddenly, especially during the course of some other disease ; the urine becomes scanty or suppressed, general dropsy develops, and œdema of the glottis may

threaten; cerebral involvement, with coma, shrill, piercing cry, and convulsions.

*Cantharis* is highly lauded by both schools of medicine. It is useful for the very acute symptoms which may arise, such as high fever, tearing pains in the kidneys, vesical tenesmus, retention of urine and uræmic coma; also in the later stages, to remove the albumin from the urine.

*Hepar*.—Urine decreased in quantity and containing blood, albumin and hyaline casts. Kafka's experience was: "No remedy will act quicker or surer than *Hepar sulph.* 3 in the case of dropsy and albuminuria occurring during scarlet fever" (*Homoopathische Therapie*). His reason for using this remedy was on the grounds of the relationship of *Hepar* to croupous inflammation.

*Lachesis* and *Terebinthina*, especially the latter in post-scarlatinal nephritis, are indicated in hæmorrhagic nephritis. In *Lachesis* the urine is very dark in color, and the characteristic subjective symptoms of the drug may be present. The urine indicating *Terebinth.* is highly albuminous and scanty, the color being "smoky," due to the abundant admixture of blood. *Helleb.* is also prominent in hæmaturia.

Uræmic convulsions call for *Cicuta*, *Bell.*, *Hyos.* or *Stramonium*; the *Arsenite of Copper* seems particularly applicable to all forms of uræmic conditions and is the remedy most to be relied upon.

All complications, such as serous effusions, œdema of the lung, etc., must be dealt with purely symptomatically. The resulting anæmia most frequently calls for *Arsen.*, *Kali carb.*, *Phosphorus*.

#### CHRONIC NEPHRITIS: BRIGHT'S DISEASE

Chronic nephritis may develop as a result of former acute conditions, especially after post-scarlatinal productive nephritis, but more commonly it complicates other important disturbances, prominently general tuberculosis; tuberculous cures; long-continued suppurative processes and hereditary syphilis. Heredity also may be an etiologic factor.

Both pathologically and clinically chronic nephritis may be classed into two distinct varieties, namely, *chronic praenchy-matous* and *chronic interstitial nephritis*.

#### CHRONIC PARENCHYMATOUS NEPHRITIS.

In this form the kidney becomes much enlarged, presenting a yellowish-white appearance (large white kidney). On section, the cortex will be found thickened and swollen and light in color, while the pyramids retain their dark-red hue.

The epithelium of the tubules is swollen and degenerated; the tubules contain degenerated cells and coagulated fibrin. Hyperplasia of the interstitial connective tissue and nuclear proliferation in the glomeruli and their capillaries, together with amyloid degeneration of the smaller vessels, are the histological lesions. Delafield sums up the pathology of the whole condition in the name he gives it, namely, chronic productive nephritis with exudation. Amyloid changes in the blood-vessels of the glomeruli are common in the nephritis of childhood.

**Symptomatology.**—As a rule, the first symptoms which will give any evidence of a renal disturbance, outside of an accidental discovery of albumin and casts in the urine, will be dropsy. The history of a former attack of acute nephritis, especially when the child is markedly anæmic and passing an insufficient quantity of urine, should, however, lead to a suspicion of Bright's disease long before the more serious symptoms make their appearance.

Anasarca usually develops suddenly, although the bagginess of the eyelids, especially in the morning, may for some time be the only manifestation. When marked, the child may become literally swollen from head to foot; with this the urine is very scanty, or even suppressed.

With the progress of the disease the quantity of urine decreases and becomes turbid from the presence of inflammatory products, urates, and sometimes blood. Albumin is found abundantly, and a microscopic examination reveals de-

generated epithelium, hyaline, granular, epithelial, and, at times, fatty casts. The specific gravity is below normal.

The *course* of the disease may extend over years; and although much more can be done for children than for adults, especially in recent and subacute cases, still the *prognosis* in a fully established case of advanced nephritis with especial involvement of the parenchyma of this important organ can offer nothing but the most unfavorable prognosis, death ultimately occurring from uræmia or some intercurrent affection (pneumonia). The exact duration is hard to predict, as the course is irregular and marked by improvement and exacerbations.



FIG. 45 CHILD THREE YEARS OLD, CHRONIC PARENCHYMATOUS NEPHRITIS WITH ANASARCA AND ASCITES.

Associated symptoms are anæmia, lassitude, indigestion, headache, and kindred nervous disturbances. Dropsical effusions into the peritoneal cavity, the pleura or pericardium may take place.

*Uræmia* is usually ushered in by severe headache and vomiting, followed by convulsions and coma. In children convulsions are more common than in adults, for obvious reasons. Uræmia is not so liable to develop when amyloid changes are marked in the kidneys, which can be suspected from the freer urinary secretion and the coexisting enlarged liver and diarrhœa.

## CHRONIC INTERSTITIAL NEPHRITIS.

This is a rare form of nephritis in children, and its etiology is not well understood. Syphilis, tuberculosis, acute alcoholism and arterio-sclerosis have been considered as causes, and in some instances it has apparently followed in the wake of an acute infectious or eruptive fever. Allan Baines (*Archives of Pediatrics*, 1901) reports a pronounced case of arterio-sclerosis with interstitial nephritis occurring in a boy ten years old. The etiology in this case was obscure excepting that he had rheumatism and chorea. Koplik cites a typical case and I have encountered it in a colored child nine years old that died of a cerebral hæmorrhage and in a boy eight years old who died of uræmia without premonitory symptoms. Guthrie has lately reported seven cases in the "Lancet." He considers it not a product of parenchymatous atrophy, but an interstitial inflammatory process with round cell infiltration of the stroma of the kidney, beginning in the cortex and spreading in the form of bands to the centre of the organ.

The *urine* is pale and abundant, low in specific gravity, and contains a small percentage of albumin, which may only be present at certain times. Such an albuminuria occurring several years after an infectious disease, the albumin being especially found in the morning urine, together with hyaline and granular casts, is a strong evidence of interstitial nephritis.

Dropsy seldom develops, but persistent gastro-intestinal symptoms and certain nervous disturbances, such as periodic headaches, vertigo, or convulsions, together with high arterial tension and hypertrophy of the heart, are indicative of contracted kidney, even in the absence of albumin. The *prognosis* depends much upon the compensation and integrity of the circulatory system, and the *course* is more protracted than in parenchymatous nephritis. Uræmia or a fatal hæmorrhage into the brain or other organ of the body usually terminates the disease.



**Treatment.**—The diet should be restricted in nitrogenous food—not, however, absolutely so, especially when there is great exhaustion and anæmia; its administration must, however, be carefully watched and entirely forbidden when uræmic symptoms threaten. Milk is the ideal food for these cases; it should hold the most prominent place in the dietary, and it can be modified in many ways to vary the monotony of its administration. Fresh vegetables, fruit and cereals, and vegetable soups made with a shin-bone stripped of the meat, or young chicken, are all allowable. There is danger of giving too much water and other fluids in these cases, as von Noorden points out. The damage done to the heart and arteries may be greater than the good accomplished by this excessive “flushing of the kidneys.”

The function of the skin should be promoted and the cutaneous circulation stimulated by the morning sponge-bath, followed by vigorous rubbing. The under-garments must be of wool, to protect against any sudden chilling.

Water should be drank regularly between meals, in moderation; such springs as Poland, Bedford and Waukesha, or a distilled water, are especially beneficial in keeping up a sufficient excretion of urinary solids.

The measures recommended for dropsy and uræmia under *acute nephritis* are equally applicable here. The remedies most useful for the nephritis itself are *Apis*, *Arsenicum*, *Aurum mur.*, *Canth.*, *Merc. cor.*, *Phos.* and *Plumbum*. These remedies are strictly homœopathic to the pathological process in the kidney, and have proven themselves of great clinical value. *Aurum* and *Plumbum* are particularly related to the interstitial form of nephritis.

If, in spite of the well-selected remedy, dropsy remains unimproved and the flow of urine dangerously scanty, nothing will act more favorably than *Apocynum cannabin.* given in ten to fifteen minim doses of the decoction (Boericke & Tafel's) in a tablespoonful of water. The action of this remedy is prompt, and I have never seen bad results from its use.



The attacks of high arterial tension occurring in interstitial nephritis are controlled by *Glonoïn*, one minim of the second decimal dilution, repeated half-hourly.

*Veratrum viride*, ix, will keep up the favorable action of the *Glonoïn*, and should be substituted for the latter as soon as the urgent symptoms have been controlled.

The nephritis consequent to chronic suppuration, vertebral caries, etc., will call for ASAF., AURUM, *Calc. phos.*, *China*, *Ferrum*, HEPAR, *Iodoform*, *Mezer.*, PHOS., PHOS. AC., *Sil.*, SULPH. The operation of stripping the kidneys of their capsule has produced a marked temporary decrease in the elimination of albumin, but it has not resulted in regeneration of the kidney parenchyma and improvement of the eliminating function as much as was hoped it might.

#### DIABETES INSIPIDUS.

Diabetes insipidus is more frequently encountered during childhood than saccharine diabetes, but both are rare diseases. Of the cases reported in literature about 20 per cent. have occurred in children under ten years.

The *etiology* and *pathology* are obscure; heredity, traumatism to the nervous system and organic brain disease, however, seem to bear distinct relationship to some cases, and it has occasionally developed after the infectious fevers.

The pathognomonic *symptoms* are polyuria and great thirst; the urine is pale and limpid, of low specific gravity, and contains neither sugar nor albumin. With this there are symptoms of impaired digestion, constipation and functional nervous disturbances. The onset is usually gradual and the course a prolonged and tedious one, either ending in recovery or in death from exhaustion or some intercurrent affection. The *prognosis* is not altogether unfavorable, especially in the young.

Differential diagnosis rests between *diabetes mellitus* and *interstitial nephritis*. From the former it is readily distinguished by the low specific gravity of the urine, the absence

of sugar, as well as lack of marked and rapid emaciation. *Interstitial nephritis* is associated with arterio-sclerosis, hypertrophy of the heart and characteristic nervous phenomena, and repeated careful examinations of the urine seldom fail to find albumin and hyaline casts. *Hysterical polyuria* is emotional in origin and only a temporary disturbance.

The remedy which has yielded the most satisfactory results in my hands in cases of persistent polyuria, when the patient has been obliged to urinate freely every two hours, or even more frequently, during the day, and four to six times during the night, the urine being pale and limpid, is *Natrum muriaticum*, 6th dilution. Thirst may be a prominent symptom, together with constipation, etc.

*Ignatia* is occasionally useful in highly nervous temperaments. Goodno has obtained positive results from *Strophanthus* 1x. Hughes recommends *Scilla* 2x; Schuessler, *Ferrum phos.* 1x. Every case will, however, require individual study, from the standpoint of the constitution, temperament and general disturbances.

#### DIABETES MELLITUS.

True diabetes is very rare during childhood, and its pathology, etiology and symptomatology need no special mention here, as it is identical with the same condition in adults. The course, however, is more rapid, and it is almost invariably fatal. The disease may terminate in a few months with diabetic coma; or if it is a mild case, yielding to treatment, it may run for years. A boy, five years old, came to my clinic, who, for the past six weeks, had lost flesh, was weak and listless and passed large quantities of urine. A specimen was immediately examined and found to contain sugar. The case was not brought back, but we learned later that two weeks after seeing him he had died. At the present time I have under my care a girl thirteen years old who has had diabetes for two years and although she is passing a large amount of sugar, and there is acetone in the urine, still her general condition remains good.

The *onset* is sudden in the majority of cases, often following upon some acute infectious disease (STERN).

The pathognomonic *symptoms* of diabetes mellitus are polyuria, voracious appetite and great thirst, with usually constipation and indigestion, marked and rapid emaciation, dryness of the skin and nervous disturbances, such as formication and neuralgia. The urine contains glucose, and its specific gravity is high. The more protracted cases frequently develop cataract. The differential diagnosis has been mentioned under *diabetes insipidus*.

**Treatment.**—The first step in the treatment must naturally be the selection of a suitable diet. The majority of clinicians adhere strictly to the diabetic diet, although quite a number recommend a more liberal mode of feeding. No doubt there are cases which thrive just as well, or even better, on a liberal diet, but personal observation leads me to believe that they are rather the exception than the rule. Goodno recommends the employment of a diet absolutely free from carbohydrates until the glucose disappears from the urine, then gradually increasing the dietary and noting the effect of each new article upon the urine. This is perhaps the most rational way of feeding diabetes, as this method withholds nothing excepting what an accurate observation has shown to be injurious to the patient. Von Noorden's method of gauging the diet in diabetes is the most accurate and scientific of all recent contributions to the literature of this subject. It is clearly set forth by Lawrence in the *N. Amer. Jour. of Hom.*, Jan., 1904. When acetone and diacetic acid are persistently present in the urine it becomes imperative to allow the patient a certain amount of *Carbohydrate*.

Meat, fish, eggs, vegetables not containing much starch, fats and oils, gluten bread and milk should constitute the diet list as far as possible. Water should be administered freely. The very best water for these cases is Allouez, from Green Bay, Wisconsin. The results from its employment are most gratifying, and Clifford Mitchell (*Hahnemannian*

*Monthly*, January, 1897) reports several remarkable cures from the use of this spring alone.

*Arsen.*—Great emaciation and exhaustion; anæmia; intense thirst; associated nephritis; complications, such as boils, gangrene, cutaneous eruptions.

*Aurum.*—Syphilitic dyscrasia; profound neurasthenia and mental depression.

*Helonias.*—Great weakness, with pain and lame feeling in back; numbness and formication in legs and feet; dejected mood.

*Lachesis.*—Development of carbuncles during the disease; last stages.

*Lactic acid.*—Gastric disturbances predominate (*Uranium nitr.*); dryness of tongue; empty feeling in epigastrium; constipation; stools hard and black; sluggish circulation in extremities. Administered in the lower dilutions.

*Lyc.*—This remedy is often indicated by its gastric symptoms, together with the presence of uric acid in the urine (*Plumbum*). Pulmonary phthisis with hectic fever.

*Nux vom.*—When the digestive tract is the main seat of disturbance; also neuropathic cases with many characteristic nervous phenomena, such as formication in the limbs; irritability; numbness and paretic condition of the lower extremities; gouty inheritance.

*Nux*, *Phosphoric acid* and *Arsenic* are perhaps the most frequently helpful remedies.

*Phos. ac.*—Cases of nervous origin. Profuse urination, with pain in back and region of kidneys, accompanied by great prostration, emaciation and sleeplessness. Rapid-growing youths.

*Uranium nitr.*—According to Prout, this remedy is especially useful when the disease originates in disturbances of the digestive tract, in contradistinction to *Phosphoric acid*, which is indicated when it originates in the nervous system.

*Rhus aromatica* is a favorite remedy with the Eclectic school, and it certainly has a marked control over the elim-

ination of sugar through the urine. It is particularly indicated when there is dribbling or incontinence of urine, being administered in doses of several drops of the tincture, three to four times daily.

#### RENAL CALCULI.

The uric acid diathesis is responsible for the majority of cases of calculi in children. These calculi are usually small and passed as gravel, inducing the characteristic pains known as *renal colic*. If not passed with the urine, they become the nucleus for a vesical calculus. Other varieties of calculus, less frequent, however, than the uric acid concretions, are those composed of oxalate of lime, carbonate of lime and cystin. Phosphatic concretions are rare during childhood, as they result from inflammatory conditions of the urinary tract, while the others are primarily found in the urine.

According to Cadge, the prevalence of renal stone in children is due to improper diet and an insufficient quantity of milk, prevailing particularly when solid or artificial foods are administered in excess. Heredity and prolonged febrile disturbances seem also to have a strong relationship to the etiology. The majority of cases have been met with among the poorer classes (SIR HENRY THOMPSON). In the newborn uric acid infarctions are normally present in the urine; they are most marked in the second half of the first day and on the second day (MORSE). No doubt they are derived from the nuclein of the leucocytes that are destroyed in the blood at this period.

**Symptomatology.**—The presence of gravel in the urine may be the only sign of any disturbance, unless pain becomes a prominent symptom. The child may cry every time it urinates, and inspection will reveal uric acid crystals and irritation of the urethra. "Occasionally, no doubt, there are renal colics quite unrecognizable in our young patients, although the urine may guide the treatment if charged with uric acid or mixed with blood" (Finlayson, *Keating's*

*Cyclopaedia*). At times, however, the typical symptoms as found in adults will be present. *Phimosis* may produce symptoms similar to those of gravel and this condition must be taken into consideration in making a diagnosis.

The symptoms due to defective elimination of uric acid are chiefly referable to the nervous system and digestive tract, making the little patient precocious, irritable and neurasthenic. Headache and insomnia are common disturbances. The digestive process is retarded, and catarrhal inflammations in various portions of the alimentary and respiratory tract frequently develop. For a fuller description of this dyscrasia see article upon "Lithæmia."

**Treatment.**—In the treatment of the diathesis the diet is of the greatest importance. Starchy foods, sugar and meat are to be avoided, encouraging the free use of milk, green vegetables and fruit. Poultry, fish and eggs may be allowed sparingly. Water should be drank freely between meals, and sufficient exercise, together with plenty of sleep, must be obtained. It used to be held that old meats were most conducive to the production of an excess of uric acid in the blood, but it has been found that the younger meats and glandular structure, such as sweetbreads, are the greatest uric acid producers on account of their richness in nuclein. The older meats, on the other hand, contain less nuclein, but they are not as digestible, owing to the larger amount of fibrous tissue they contain.

For the constitutional condition, one of the follow remedies will usually be indicated:

*Berberis*.—Yellow turbid urine, mushy sediment.

*China* —Urine is pale, becoming turbid on standing, with yellowish flocculent sediment, or scanty urine with brick-dust deposit.

*Lycopodium*.—Brick-dust deposit in urine. It is scanty, high-colored, and stains the diaper a deep yellow. Whenever the child urinates it cries from the burning and smarting produced by the irritating urine.

*Nux vom.*.—Gastric symptoms ; constipation ; insufficient exercise and sleep ; gouty inheritance ; painful urging to urinate ; pain in right kidney, worse lying on the affected side ; reddish urine.

*Natr. mur.*.—Profuse and frequent urination. The urine is pale, and deposits a brick-dust sediment. Constipation ; emaciation ; anæmia.

*Sepia.*.—Urine offensive, with greasy pellicle, leaving a pink, paint-like deposit in vessel.

*Sulphur.*.—This remedy is frequently indicated by its general characteristics.

For the painful symptoms, one of these remedies should be considered: *Arg. nitr.*, *Acon.*, *Arsen.*, *Bell.*, *Berb. vulg.*, *Canth.*, *Dioscorea*, *Lyc.*, *Nux vom.*, *Pareira brava*, *Tabacum*, *Uva ursi*. The inhalation of an anæsthetic or the administration of a narcotic is justifiable in the presence of uncontrollable, excruciating pain.

*Berberis.*.—"I wish to sing the praises of *Berberis* as a general remedy for pains centering in the region of the kidneys, radiating thence in every direction, especially down the ureters."—(WM. BOERICKE. *North Amer. Jour. of Hom.*, May, 1898.)

#### CYSTITIS.

While cystitis is not a common affection of childhood, still there are certain conditions that frequently lead to its development. Vesical calculus, for example, although not invariably setting up a cystitis, is one of its commonest causes. In this connection it will be well to study the symptoms of stone in the bladder as detailed below, for there are certain deviations from the symptom-group which is found in adults. The acute infections, notably scarlet fever, diphtheria and typhoid fever, are at times complicated with acute cystitis, usually as a result of infection following retention. The *bacillus coli communis* is credited with being the cause of the majority of cases of cystitis, although the typhoid and diphtheria bacillus



may act as the exciting agent. Besides these varieties there is the *tuberculous cystitis*, and in rare instances the *gonococcus* has been known to set up cystitis in children (BAGINSKY).

*Retention of urine*, either during the course of an infectious disease or from phimosis or other congenital narrowing of the urinary tract, is a strong predisposing cause.

Errors in diet leading to *oxaluria* may be the cause of frequent and painful urination, but the condition can hardly be termed cystitis. The same is true of *lithæmia*.

Among the rarer causes are *tumors* and *foreign bodies* in the bladder.

Girls are more often affected than boys.

**Symptomatology.**—In *acute* cases there is fever and restlessness; tenderness over the bladder; frequent and painful urination, the urine containing mucus, pus, and usually blood.

In the *subacute* and *chronic* forms the symptoms are less intense and there is generally no fever. Here it becomes necessary to determine whether it be a simple cystitis, a tuberculous cystitis, or a case of stone in the bladder. Nephritis must also be excluded.

*Simple cystitis* is not associated with pronounced hæmaturia and seldom assumes a prolonged and chronic course under appropriate treatment. The urine contains mucus in abundance, together with bladder epithelium and pus corpuscles. The cystitis can usually be traced back to an acute infectious disease or to an attack of retention from phimosis or other cause.

*Tuberculous cystitis* is associated with much pain, and hæmaturia is an early and prominent symptom. The urinary sediment is less gelatinous and more flocculent than in simple cystitis (TERRILLON). It usually retains its acid reaction. By rectal examination enlarged lymphatics may be felt, and there may be glandular swelling in the iliac fossa (ASHBY and WRIGHT.) Lastly, the presence of the *tubercle bacillus* in the urine will render the diagnosis absolute.

An *irritable bladder*, the result of some form of irritation in the genito-urinary tract, most commonly phimosis, lithæmia, or phosphaturia, is recognized by the entire absence of any inflammatory condition.

*Stone in the bladder* may set up a severe cystitis, but this is not an invariable result of calculus. There may be nothing more than vesical irritability. Stone is most commonly of the uric acid variety, its source being the kidney. The classical symptoms of stone observed in the adult may be wanting, the most prominent symptoms of this condition in children being extreme vesical tenesmus often leading to *prolapsus ani*. Any case of prolapsus ani of long standing should lead to an examination for stone.

**Treatment.**—Rest in bed is imperative in the acute form. When the acute symptoms have been controlled, should there still remain an excessive amount of mucus and some pus in the urine, the bladder should be washed out daily with a two per cent. solution of boric acid. All mechanical conditions, such as phimosis or calculus, must receive appropriate surgical treatment. A liquid diet, consisting mainly of milk and water in large quantities, is imperative in acute cases; chronic cases should avoid meat in excess; also stimulants and acids excepting the fruit acids. An alkaline water is of great benefit, while urinary antiseptics are sometimes useful. *Boric acid*, grains one to three, three times daily, and *Urotropin* in the same dosage are the ones to be recommended. Of internal remedies, *Cantharis* is no doubt the most useful. In acute febrile cases, *Aconite* or *Belladonna* may be indicated. In chronic cases, *Lycopodium* is very important. Other remedies to be considered are: *Apis*, *Berberis*, *Hyoscyamus*, *Terebinthina*, *Uva ursi*, acute cases; *Chimaphila*, *Dulcamara*, *Hydrastis*, *Lycop.*, *Natr. mur.*, chronic cases; *Arsenicum*, *Arsenic. jod.*, *Lycop.*, *Sulphur*, tuberculous cases.

## ENURESIS.

Enuresis cannot be said to exist as a pathological condition until after the second year, for the child does not learn to voluntarily hold back the urine until after the first dentition period. A lack of physiological development of the sphincter vesicæ or an excessive irritability of the bladder are the essential features of this neurosis, although reflex irritation frequently seems capable of inducing enuresis in many instances. In the majority of cases, however, both of these conditions are operative, and, accordingly, both must be corrected before a cure can be established. Bruck (*Der Kinderarzt*, Feb., 1900) expresses the belief that the heavy sleep natural to children is one of the chief predisposing causes to enuresis. It is more common in boys than in girls.

Enuresis exists primarily in children who are anæmic, neurasthenic or otherwise poorly nourished, and especially in nervous temperaments, the precocious being perhaps most afflicted, although the idiotic are late in learning to control micturition. Symptomatically it may occur in almost any organic nervous disease, particularly in epilepsy, where nocturnal enuresis is often the earliest symptom attracting attention, the convulsive seizure having been overlooked.

A highly-acid urine, cystitis and vesical calculus are also prominent causes of this affection.

Among the reflex disturbances capable of exciting enuresis must be considered phimosis; adherent prepuce and clitoris; abnormally small meatus; adenoid vegetations; rectal fissures and polypi; seat-worms.

Habit must also be credited often as playing a prominent rôle.

**Symptomatology.**—Wetting the bed is the most frequent form of enuresis, but in many instances the child is unable to control the urine during the day as well as night. Rarely is it purely diurnal. There is no dribbling, but the mere thought of urinating induces contraction of the walls of the

bladder, the force of which the sphincter is unable to overcome. During the night a dream may suggest the idea of urinating, with consequent wetting of the bed, or the act may occur purely reflexly. In the majority of cases the involuntary micturition occurs in the first hours of the night—a time when sleep is usually most profound.

The *course* is variable and depends entirely upon the cause. In sound children with enuresis depending purely upon reflex irritation a cure follows promptly upon the removal of the cause. Again, I have seen prompt cure follow when systematic exercise and a cold morning plunge bath was prescribed, after drugs had failed. In feeble, neurotic or degenerate children it may prove most stubborn and protracted. Of course, almost any case can be controlled, at least temporarily, by the use of atropine, but this is not a desirable procedure and should only be resorted to when the long-continued bed-wetting threatens to demoralize the child through shame and loss of self-control. Enuresis rarely persists after puberty.

**Treatment.**—When beginning the treatment of a case of enuresis the physician should bear in mind that this is only a *symptom*, the cause of which he must seek to fathom and remove. Anatomical defects must be corrected by surgical means, and lithæmia, oxaluria and phosphaturia, seat-worms, rectal fissures, etc., must receive their just share of attention before the enuresis can be properly treated.

The nose and throat must be thoroughly inspected and pathological conditions here receive appropriate treatment. Let us study the state of the nutrition and decide whether the child is getting sufficient food, exercise and fresh air. If the child is not too anæmic and the heart is normal a cold plunge bath may be taken in the morning. When this might seem too severe the sponge-bath may be substituted.

The bed clothes should not be too warm and fluids should be taken in small quantities in the evening. It is a good plan to waken the child about the time urinating usually

takes place and thus get it away from the habit of doing this involuntarily. Elevating the foot of the bed has been useful in some cases. From the various specifics so highly praised I have not obtained uniform results; they, too, must suit the case in every respect, as well as any other remedy, in order to be curative.

*Acon.*—Recommended in cases of neurotic origin; child awakens from sleep in fright; *feverishness, due to seat-worms.*

*Bell.*—This is the specific, usually employed in the form of *Atropine*. When indicated by its characteristic nervous symptoms it frequently relieves in potency. Its action upon the involuntary muscle fibre of the bladder in large doses is a paralyzing one, in this way controlling the over-sensitive organ.

*Benzoic acid.*—Strong penetrating urine. Dilute *Nitro-muriatic acid* is useful in highly lithæmic cases.

*Caut.*—Enuresis during first sleep; atony of the sphincter vesicæ. Hughes has often used it with success.

*Cina.*—Helminthiasis.

*Equisetum.*—This remedy has quite a reputation in enuresis, being employed in drop-doses of the tincture. It seems to control the habit very satisfactorily in many instances.

*Ignatia.*—*Ignatia* has given me the best results in those nervous, irritable children who are precocious and neurasthenic, and in whom the condition is due to a hyperæsthesia of the neck of the bladder and urethra. This can be demonstrated by the passage of a sound, which induces intense burning pain out of all proportion to the usual discomfort accompanying this operation. Curative results were obtained by the use of *Ignatia* alone, although passing cold steel sounds is considered highly beneficial, especially in young boys who masturbate (POWERS, *Surgical Diseases of Children*). For the prostatic irritation induced by this vicious habit there is no more useful remedy than *Staphisagria*.

*Ferrum* and *Ferrum phos.* have proven beneficial in anæmic children; they are recommended for the diurnal variety.

*Sulphur*.—"The remedy which, among all others, has given me the quickest results is, without any doubt, *Sulphur*, no matter if the children were blonde or brunette, fat or thin, etc." (JAHR, *Therapeutische Leitsfaden*). He recommends this remedy to be given first in every case in the absence of strong indications for another. In my hands it has only been of service when the typical sulphur constitution was present, but it is, no doubt, one of our most valuable remedies in enuresis. The child requiring *Sulphur* is lithæmic or neurasthenic. It is fond of sweets and highly seasoned foods and complains of a host of nervous and dyspeptic symptoms too numerous to mention.

Besides these remedies the *Calcareas*, *Lyc.*, *Plantago*, *Puls.* and *Sepia* have been recommended.

#### VULVOVAGINITIS; GONORRHŒA.

Vulvovaginitis is a catarrhal inflammation of the mucous membrane of the vulva and adjacent parts and in the cases encountered in hospital and dispensary practice is most frequently gonorrhœal in origin. Anyone who will take the trouble to examine the pus from these cases will find to his surprise diplococci within the pus cells staining distinctly with aniline dyes and decolorizing by Gram's method (the *gonococcus of Neisser*). Koplik (1893) cultivated the organism, definitely proving its identity. This has been done since repeatedly.

By no means every case of vulvovaginitis is gonorrhœal, however, but the proportion of specific cases is so constant that the subject of gonorrhœa, its complications and its control in children, has become one of the most important topics in pediatrics in late years.

*Non-specific vulvovaginitis* is a simple catarrhal process due to lack of cleanliness; local irritation, such as smegma, seat-worms or masturbation; or it may be but part of a general catarrhal condition in scrofulous children.

There is a form of purulent vulvovaginitis and urethritis



affecting both male and female children which is distinctly contagious and is due to a diplococcus which does not show, however, the staining and cultured peculiarities of the gonococcus (KOPLIK). It is conceivable that this is a degenerate or attenuated form of the gonococcus.

The *spread* of vulvovaginitis is surprisingly sure and rapid when children are brought into close contact, as in a hospital, for example. Every precaution should be taken, therefore, to prevent contagion.

Many children contract the disease from their mothers or attendants. It is often difficult to find the original source of infection. Rarer modes of transmission are by rape and attempted sexual intercourse of young boys with other children or women. Such cases we occasionally see in the dispensaries.

The gravest aspect of gonorrhœa is its *complications*. Salpingitis and peritonitis have been observed (MARX; SANGER). This leads to death or sterility. It is a rare complication. Infection of the eyes—ophthalmia—is constantly to be dreaded.

*Arthritis* in children is not infrequently gonorrhœal. Holt and Kerley have observed that the majority of arthritides that were formerly looked upon as being septic are gonorrhœal. Kimball (*N. Y. Med. Record*, Nov. 20, 1903) reports eight cases of pyæmia with joint involvements in infants in all of which the gonococcus was demonstrated. No primary local lesion was present. The majority died during the height of the attack.

One of my cases, an infant three weeks old delivered in the Hahnemann Hospital Maternity developed ophthalmia three days after birth and a week later successive involvement of the shoulder, elbow and hip-joints. On one hand the second finger became involved in a fusiform swelling (dactylitis) and the wrist was also swollen. The temperature was continuously elevated, ranging from 101 to 102.5° F. Dr. Sappington obtained pus from the joints in which he demonstrated gonococci, verified by cultures on ascitic



fluid and agar. The child was taken home when four weeks old and died shortly after from marasmus, after apparent improvement in the joint condition.

**Treatment.**—In the acute stage the local condition is much benefited by irrigation with a warm solution of a non-irritating silver preparation. One pint of a 1 to 500 solution of *Protorgol* or *Albargin* (I prefer the latter) may be used twice daily. The vulva and vagina are most satisfactorily irrigated through a small, soft rubber catheter, which may be gradually introduced into the vagina as the secretion from the external parts is flushed away. The vulva is then dried and dusted over with *Boric acid*.

In the subacute and chronic stage *Permanganate of Potash* 1-1,000, may be used every day or two in a similar manner followed by the dusting powder. Sometimes the dry treatment will give better results than douches.

In the early stages *Cannabis Indica* is indicated, or *Cannabis* when there is dysuria. When the discharge becomes profuse and yellow *Pulsatilla* is the most useful remedy. In chronic cases, *Sulphur* or *Sepia*.

Non-specific vulvovaginitis usually calls for *Calc. carb.*

## CHAPTER XIV.

### DISEASES OF THE SKIN.

Certain peculiarities characterize the condition of the skin in the new-born. At birth the entire body is covered with a waxy secretion, the vernix caseosa, which has served as a protective layer to the skin during intra-uterine life. The color is a deep red, owing to the vigorous surface circulation, and this condition persists for about a month. Usually desquamation of the epidermis, visible on close inspection, takes place during the second week. Jaundice, occurring on the third or fourth day, was found in 80 per cent. of all infants observed by Runge.

The skin of the infant is exceedingly tender and susceptible to all forms of local irritation, as well as being readily disturbed in its function and structure by abnormal conditions. Sweating is rare in infants, normally not noticed before the fourth month, and when persistent it becomes a strong presumptive sign of rickets. The use of irritating soaps, excessive bathing, and, on the other hand, uncleanness, are important etiological factors in the skin diseases of childhood, next to which improper feeding ranks. Syphilis and parasites are other prominent causes.

Almost any of the skin diseases of adults may be encountered in children, but the ones here described are the most common and important.

In all instances the cause of the disease, if possible, must be removed. Diseases due entirely to local irritation and those responsible for their existence to parasites rarely demand anything but local treatment, while those dependent upon a hereditary taint or constitutional dyscrasia must be eradicated by the administration of the well-selected homœopathic remedy.

## INFLAMMATIONS: ECZEMA; TETTER.

**Definition.**—Eczema is an inflammatory, acute or chronic non-contagious disease of the skin, in the beginning presenting erythema, papules, vesicles or pustules, often in combination, associated with a varying degree of infiltration, burning and itching, and ending in serous and puriform degeneration. The formation of scales and crusts is a usual sequel.

**Symptomatology.**—Any or all varieties of eczema may be present in infancy and childhood. *Eczema erythematosum* appears primarily as a reddened, mottled condition, without exudation. Later the involved surfaces may become excoriated and throw off a few scales of epidermis. In children it is seen most frequently about the genitals, the buttocks, and between the thighs, as a result of maceration of the epidermis. This form of eczema, or *intertrigo*, as it is frequently called, is a common and troublesome condition showing strong tendency to relapse when the infant is not cared for most tenderly.

*Eczema papulosum.*—This variety presents papules of varying size, surrounded by an erythematous or empurpled base and surmounted by a layer of thin scales. From incessant scratching the summit of these lesions becomes abraded and excretes a sticky serum, producing an *eczema vesiculosum*. The trunk and flexor surfaces of the limb are usually involved. This variety is slow to respond to treatment.

*Eczema vesiculosum.*—Vesicles, usually minute, characterize this type. Their apices are filled with a yellowish, sticky fluid. In most cases they rupture and coalesce, forming crusts. The lesions are usually situated upon the face, neck and scalp, and are attended with severe burning and itching. The vesicular variety is common to infants.

*Eczema pustulosum.*—In some cases, either because of a peculiarly favorable soil or by reason of the intensity of the inflammatory process, pustules rapidly develop upon a group of papulo-vesicular lesions. Indeed, it is not uncommon to find a lesion commencing as an erythema and running

through all of the stages to pus formation. Eczema pustulosum usually results from a distinct pyogenic infection, traceable to scratching with dirty finger-nails. *Eczema rubrum* is not a distinct variety. It is a name applied to a condition presenting a complexity of symptoms, including erythema, papules, vesicles, pustules and scales. The parts involved are reddened, infiltrated, excoriated, and frequently covered with crusts. It is usually found about the bends of joints, and is attended with marked discomfort.

*Eczema squamosum*.—This is a chronic variety, and usually results from an attack of erythematous or papular eczema. Typical cases show thickened and infiltrated areas, situated upon the face and back of the neck. Sometimes the lesions are widely scattered.

**Etiology.** - Of late, dermatologists have attempted to establish the parasitic theory of eczema, but have failed because of inability to discover a specific micro-organism. Scabies and pediculosis are often responsible for an eczema, the condition resulting from the irritation and scratching these parasites induce. The disease in infants may be traced directly to the action of local causes; particularly is this true with the newly-born, who are subjected to vigorous baths and energetically anointed with irritating lard and afterwards tightly enveloped in woolen garments—procedures likely to irritate a tender and delicate skin. The irritating discharges accompanying the diarrhoea of infancy frequently produce a local dermatitis, which is quickly converted into a weeping eczema. A tight or elongated foreskin and catarrhal inflammation of the vulvo-vaginal glands are frequently responsible for eczema in these parts. The too liberal application of water, the use of impure soaps and toilet articles, tight and heavy clothing, may provoke an attack. Vaccination plays a rôle in the causation of eczema. From a clinical standpoint, dentition and eczema seem to be closely related; and, although it is difficult to understand this, yet attacks are aggravated by the eruption of a tooth, subside shortly after-

wards, and occur upon the eruption of others. Occasionally too liberal indulgence in sweets will excite an attack. The fundamental cause of eczema of infants and children may be traced to a constitutional diathesis, usually hereditary, sometimes acquired. Eczematous parents often beget eczematous children. Although eczema occurs among the children of the rich, it is seen most frequently amongst the poor and ill-nourished, particularly amongst light-haired children, who show a tendency toward catarrhal affections of the upper air-passages (the so-called "scrofulous" diathesis). Eczema is occasionally associated with impetigo contagiosa and some of the other pustular dermatoses; it may follow scarlatina and varicella.

An attack may assume an acute, subacute or chronic form; the majority of cases are subacute. A varying degree of burning and tingling accompanies all phases of the disease. Acute infantile eczema is accompanied by moderate fever; the skin of the affected region assumes a reddened hue, and is attended by catarrhal exudation. Itching and burning are prominent symptoms. Acute infantile eczema usually commences upon the scalp, a favorable soil being furnished by the sebaceous secretions of this region. This, together with the gummy contents of the vesicles, forms dirty, grayish-yellowish crusts (*crusta lactea*).

Through infection or the association of head lice this condition may become most repulsive and malodorous. In advanced cases the glands about the ears and neck become involved, and not infrequently suppurate. Furunculosis of the scalp, face and neck is by no means a rare complication. Itching, scratching and inflammation reduce the sufferer to a condition often grave.

In scrofulous children a pustular condition of the eyebrows results in subsequent crusting and destruction of the hair-follicles; frequently catarrhal conjunctivitis is present. The mucous membrane of the nose becomes involved. At first a thin, watery, later a thick, purulent discharge occurs, which

forms pustules about the lips and angles of the mouth, resembling very much the lesions of *impetigo contagiosa*. Eczema genitalium frequently complicates the condition, presenting an erythematous variety, which may assume a vesicular form and invade the spaces between the thighs. The perinæum, anus, penis, scrotum and labia may become affected. Under such circumstances the parts assume a scarlet hue, becoming swollen, infiltrated and raw; vesicles predominate.

The chronic eczemas of infancy and childhood are confined mostly to the scalp, face, and naso-labial furrows, rarely to the prepuce and anus. A chronic eczema capitis is usually confined to a small area about the occiput. Here papulo-squamous lesions predominate; sometimes a pustular type is found, for which parasiticism is largely responsible. Chronic eczema of the nose and angles of the mouth present fissures, crusts and shallow ulcers. A similar condition is seen in chronic eczemas about the genitals and anus.

**Pathology.**—In acute eczema the pathological changes may be diffuse or circumscribed. They are primarily situated in the papillary portion of the derma, although they may later descend as deep as the fatty layer. They consist of congestion of the blood and lymph vessels, causing a serous exudate, which gives rise to infiltration and induration of the skin. This exudate may destroy the rete cells; the formation of vesicles or bullæ may be entirely absent, and the exudative inflammation result only in the destruction of the epidermis. In chronic eczema the inflammatory changes are most marked about the blood-vessels in the derma; the papillæ become hypertrophied; proliferation of connective-tissue cells takes place; the subcutaneous tissues may become infiltrated and thickening result.

**Diagnosis.**—Certain characteristic symptoms are invariably present in eczema; burning and itching are peculiar to eczematous eruptions, and the skin is usually infiltrated. Some discharge may usually be seen; it may be colorless or

stain the clothing. Since eczema simulates so many diseases of the skin, it will be necessary to point out a few diseases for which it may be mistaken.

*Erysipelas* is an acute inflammation, involving the deeper layers of the skin. It commences from a single focus and spreads rapidly, while eczema starts from a larger area. There is more fever in erysipelas, and, as a rule, no discharge. Erysipelas is rare in infancy, excepting in the new-born, as a result of septic infection (see "Diseases of the New-Born," p. 115).

*Psoriasis* is rarely seen in infants and very young children. A psoriatic condition of the scalp may suggest a squamous eczema of that region. The patch of psoriasis, however, is sharply defined, and the scales are abundant, large and silvery. Psoriasis is always dry, while eczema usually presents some degree of moisture. Psoriasis selects the extensor and eczema the flexor surfaces of the limbs.

*Urticaria* may be mistaken for a papular eczema, but the lesions of urticaria are wheals; the disease usually follows acute indigestion, and is of short duration. There is, however, a form of urticaria common in children, described as *vesicular urticaria*. Here small papules and vesicles develop over the body generally, especially on the extremities, and they are more persistent than the ordinary form of urticaria. They are, however, discreet lesions and tend to spontaneous cure.

Papular eczema presents certain features peculiar to *lichen ruber planus*; both eruptions itch; both present papules. The lichen papule, however, runs a chronic course, does not change its identity, and always leaves a certain amount of pigmentation behind.

*Syphilis*.—In all cases where the slightest possibility exists of a skin eruption being syphilitic we must inquire most thoroughly into the previous history of the case as to snuffles; persisting hoarseness; papules, pustules and ulcerating lesions about the anus and genitals. The mother's history



must also be inquired into, especially as to miscarriages. Usually syphilitic eruptions are characteristic—their color, distribution and polymorphism being pathognomonic. In chronic relapsing syphilides, for instance, the circinate papular syphiloderm, the diagnosis is not always easily made, but their location (forehead), circular outline and distinct scaliness with absence of itching should serve to differentiate them from eczema.

The papules of syphilis do not itch and are usually grouped. An eczema of the scalp may resemble syphilis of that region, particularly where the lesions are of a pustular character. The syphilitic process, however, is more extensive; the disease makes inroads upon the scalp and ulceration is somewhat extensive; rupial crusts are usually present. As a last form of evidence, the therapeutic test may be applied.

The points of resemblance which *impetigo contagiosa*, *pediculosis capitis* and *scabies* bear to eczema are pointed out under these special subjects.

**Prognosis.**—The course of an eczema depends upon so many circumstances that it is difficult to accurately foretell its outcome. As a rule, it runs a chronic course—age, the location affected, the exciting causes, heredity and constitutional predisposition are factors which must be considered. In the majority of cases the existing eruption can be controlled. Proper hygienic surroundings and cleanliness will cure many of the simpler types, while those dependent on a scrofulous taint are exceedingly rebellious to treatment. Acute eczema capitis, if uncomplicated by parasiticism, may, by proper treatment, be quickly controlled. Eczema of the nose is slow to respond to treatment. The eczema of the genitals and anus are peculiarly hard to control, while the squamous and papular types often persist for years.

**Treatment.**—It would be impossible to decide in a general way the treatment of eczema in infants and children. Each case, each type and each variety must be isolated and treated as a distinct affection. An internal remedy given for an ec-

eczema of the scalp, caused by pediculi or other parasites, would be a waste of time and labor. On the other hand, an eczema caused by a psoric taint will never yield permanently to anything but a constitutional remedy.

Piffard (*Morrow's System*) is a firm believer in the efficacy of internal medication in eczema and recommends as the most useful drugs *Arsenic*, the *Sulphide of Lime* (*Hepar sulph. calc.*) and *Viola tricolor*. The indications given are mainly those upon which we, as homœopathists, base our prescriptions.

In every case the cause must be sought for, and, if possible, removed. Its effects may then be rationally eradicated. The question should be asked: "Is this condition due to a local or constitutional cause, or are both agents responsible?" Again, there are regional eczemas which demand peculiar measures for their relief. An inquiry into the causes of the vast majority of cases occurring in infants elicits a history of a psoric taint. Internal medication is usually alone indicated. In those cases arising from stomach or intestinal derangement, caused by an unsuitable food or overfeeding, a proper diet must be prescribed, which, in many cases combined with an internal remedy, will effect a cure.

If the infant be a suckling, the mother's milk should be analyzed and if the fat or proteid percentage be too high, her diet must be so regulated as to correct this fault (see Chapter on Infant Feeding). The same applies to artificially fed infants, in whom the diet must be carefully regulated and the percentages of proximate principles in the food made to conform with the state of the digestion and nutrition. In older children the usual dietetic error is eating too much starchy food and this should be prohibited. Water is to be given freely and constipation carefully guarded against.

The local treatment will depend solely on the type present. In every instance, however, we must first decide: "Does this eruption demand a stimulant or a sedative?" Usually acute cases demand sedatives, while chronic ones call for stimulants.

Acute eczema erythematosum is usually controlled by the following simple dusting powder :

R Pulv. amyli, . . . . .	3 <i>ij</i> .
Zinci oxid., . . . . .	3 <i>iss</i> .
Pulv. camphoræ, . . . . .	3 <i>ss</i> .

This should be applied to the parts several times daily. It is particularly serviceable in controlling the itching. Where the genitals become affected, *Subnitrate of Bismuth*, alone or combined with *Starch*, *Talcum* or *Lycopodium powder*, may be applied with beneficial results.

The parts should not be bathed in water, but should be mopped with a soft woollen rag and bathed with pure olive oil. After this any of the above powders may be used, and a thin layer of linen placed between the thighs. The internal remedies best suited for such cases are *Aconite*, *Belladonna* and *Mercurius*. *Pulsatilla* should be given to those cases affected by gastric derangement.

Vesicular eczema is usually found as a subacute or chronic condition. Frequently pediculi are present, especially when the scalp is invaded, and they should be exterminated promptly. Scales and crusts may be removed by soaking thoroughly in olive oil. These must be entirely removed before a cure may be hoped for ; after which a mild antiseptic powder should be used several times daily (*Boric acid* diluted with two parts *Starch* or *Zinc oxid.*). Intense itching is very frequently controlled by the use of a very weak solution of *Carbolic acid*, five drops to the half-ounce of water. Internally, a remedy of most positive value in the acute vesicular stage is *Rhus tox.*; its proving shows a vesicular eruption, attended with itching, burning and tingling.

The pustular stage demands *Hepar*; later, when the pustules rupture and throw out a thick yellow fluid, which quickly dries and forms crusts, *Graphites* will be needed ; and occasionally, where these symptoms are present and the glands of the neck become involved, *Sulphur* may be advantageously employed. Frequently, in marasmatic children,

acute eczema capitis assumes a chronic type; the lesions are squamous, surrounded by an inflammatory base; the hair is dry, lustreless and brittle; the scalp bleeds easily; here *Sulphur* and *Arsenicum* meet the conditions, while *Calcaria carbonica* is indicated in pale, fat, flabby and pot-bellied children. Occasionally local remedies are of value, but not nearly as much so as in the acute variety. The main indication for local treatment is to keep the parts absolutely clean. As plain water irritates, a two per cent. solution of *Boric acid* may be used. Should a stimulating application be necessary to hasten the cure, a five per cent. ointment of *Ichthyol* may be used.

Acute vesicular eczema of the face calls for *Rhus tox.* *Apis* is indicated if much œdema and erythema are present *Graphites*, where the condition becomes chronic. The itching in these cases is sometimes intolerable and makes it necessary to apply a mask of soft muslin with a layer of *Zinc ointment* and *Lanolin*, equal parts, as a protective. The child's hands should be encased in mittens; often it is necessary to restrain the arms entirely by pinning them to the sides of the body with a sheet wrapped around the body. The best anti-pruritic washes are *Boric acid*, *Resorcin* and *Carbolic acid*, one to two per cent. solutions of each.

Eczema of the eyes and of the border of the lids should be treated by washing with a saturated solution of *Boric acid*. This dissolves the crusts, and is beneficial to the accompanying conjunctivitis. This variety occurs in badly-nourished, scrofulous children, and calls for the employment of the following remedies: *Graphites*, *Sulphur*, *Calcaria*.

One of the most obstinate forms in infants is eczema of the genitals and surrounding structures; it may assume any clinical variety. It is usually acute, but may be chronic. The surface is raw, and usually exudes a sticky fluid; the genitals become swollen. The inflammation is best combated by protecting the skin from the contact of the urine and fæces with *Calendula cerate*. In some cases a cerate is not

well borne and a dusting powder will act better (*Boric acid*, *Zinc oxide* and *starch*, equal parts). Internally, *Aconite*, *Arnica*, *Bryonia*, *Rhus tox.* and *Graphites* are indicated. The subacute types demand a recognition of their cause and prompt removal; in some cases a long foreskin, by causing frequent urination, may cause an eczema. Here, after the control of the acute symptoms, circumcision is demanded. In most cases it must be remembered that diet and hygiene will do much toward a cure.

#### ERYTHEMA.

Erythema may be defined as a redness of the skin which disappears temporarily upon pressure. Clinically two groups are recognized, erythema simplex and erythema exudativum. Erythema simplex presents a number of types, among which are erythema traumaticum, erythema caloricum, erythema venenatum and erythema intertrigo, and the different forms of symptomatic erythema, all arising from various causes.

*Erythema simplex* is characterized by an eruption of reddish macules of varying size, which disappear upon pressure. The causes may be internal and external, the condition arising from friction, brought about by wearing tight clothing, or from the action of an external irritant. Extremes of temperature are responsible, and, in some cases, reflex vaso-motor irritation, or the ingestion of certain articles of food or drink.

A more diffuse erythematous rash not infrequently occurs during attacks of indigestion as a result of auto-intoxication. An erythema of wide distribution may also precede the appearance of the papular rash in small-pox or it may accompany some of the non-eruptive infectious diseases (typhoid fever, diphtheria, tonsillitis). The same may be observed after vaccination. Any portion of the surface of the body may be invaded. The lesions may be widely scattered. Fresh crops are usually bright red, fading as they become older. Occasionally a slight degree of pigmentation may remain. Itching, burning and tingling, and, in some cases, more or less elevation of temperature may be present.

*Erythema traumaticum*.—This results from external irritation, and, like the former variety, is also due to wearing too tight clothing, and may be produced by too vigorous friction after the bath. It rapidly subsides after the removal of the exciting cause, but may, in some cases, result in acute eczema.

*Erythema caloricum* results from the action of extremes of temperature, very low temperature or the application of ice may produce a diffuse redness which, if continued, may cause a dermatitis; high temperature, particularly exposure to the sun's rays, will cause an erythema which may be brief, or which, in severe cases, may terminate in a vesicular eruption.

*Erythema venenatum* may be traced to the application of certain irritating substances, such as mustard, pepper, turpentine and ammonia.

*Erythema intertrigo* is a redness of the skin at points where natural folds come in contact, as the neck, armpits and thighs. It is common to infants possessing a delicate skin, and, unless promptly recognized and quickly eradicated, may terminate in an eczema madidans. Usually there is burning, itching and tingling, and a certain amount of exudation.

*Erythema scarlatinoides*.—The clinical importance of this form of erythema rests upon its close resemblance to scarlet fever. Although the majority of cases are mild and evanescent in character, still there are such in which the entire body is covered with rash in association with fever and grave constitutional symptoms.

The *etiology* is most frequently to be found in some form of toxæmia; the commonest source of this is the intestinal tract. Eating certain food (shell-fish) or tainted meat may give rise to an attack. It is sometimes associated with certain infectious diseases and with sepsis. *Mercury* and *Iodoform* have also produced similar symptoms.

The *eruption* usually appears suddenly, although it may be preceded by headache, malaise and fever. The lesions are mostly confined to the face, neck, trunk and extremities.



They are macules or papules of a bright-red color, which generally coalesce. It may be difficult to differentiate this variety from scarlatina, particularly during the first twenty-four hours. Usually, however, the eruption quickly fades, leaving none of the grave symptoms attending scarlet fever. Burning and itching may be annoying symptoms. Desquamation is marked in most cases and recurrences are common.

The *diagnosis* from scarlatina rests upon the absence of exposure to contagion; the less general distribution of the rash and absence of strawberry tongue, sore throat and adenopathy; absence of albuminuria.

*Erythema medicamentosa.*—This type, like the foregoing, usually follows the ingestion of drugs. The eruption is macular, and in some instances papulo-vesicular, and even pustular. Rarely it may be scarlatiniform in character. The eruption is scattered over the head, trunk and limbs, and disappears upon the removal of the exciting cause.

*Treatment.*—*Erythema simplex* is readily controlled. All sources of local irritation must be removed and the diet strictly supervised. Internally *Nux vomica*, *Pulsatilla*, *Hepar*, *Ipecac* and *Bryonia* may be indicated. Local irritation may be subdued by dusting powders, and if itching is severe a bran-bath will be of service. *Erythema intertrigo*, if not promptly controlled, may result in a very acute eczema. Absolute cleanliness must be observed and all irritants removed. After thoroughly bathing, the parts should be dried with a soft towel and freely dusted with equal parts of *Starch*, *Zinc oxide* and *Boric acid*. Internally *Aconite* and *Belladonna* may be administered very early in an attack; persistent diffuse redness is relieved by *Mercurius*, and where vesicles form *Rhus tox.* should be given.

#### FURUNCULOSIS; BOILS.

A *furunculus*, or *boil*, is an acute, deep-seated, circumscribed inflammation originating in a hair follicle or sebaceous gland and terminating in necrosis of these structures and surround-



ing connective tissue. The *cause* is infection with *staphylococcus pyogenes aureus*.

**Symptomatology.**—Slight itching and burning, associated with a moderate degree of localized infiltration, marks the site of a coming "boil." Within a day or two a conical papulo-vesicle appears, which later becomes filled with pus. This pustule is surrounded by a markedly infiltrated base, and there is considerable elevation of the skin. Where the deeper structures are involved, the skin becomes thinned and assumes a bluish hue. Within a few days the tumor may have attained a considerable size. Intense throbbing pain, made worse by motion, adds greatly to the patient's discomfort. Central coagulation necrosis quickly takes place, resulting in the formation of a "core," or what is more common in children, a small localized abscess. After an opening is formed the pain and fever rapidly abate, and the opening quickly fills up with granulations. Deposits of pigment may persist for some time. Successive crops appear from time to time, extending over a period of months. This condition is termed furunculosis, for which a constitutional cause is frequently responsible. Boils are usually seen upon the neck, back and nates.

Furunculosis frequently accompanies and often follows an attack of scabies or pediculosis. This will be readily understood by recognizing how irritated and inflamed the skin becomes as a result of the incessant scratching accompanying these parasitic diseases, thus inviting the entrance of pyogenic organisms. Improper and tight clothing, irritating soaps, poultices, and the too lavish use of strong antiseptic lotions may be contributing factors; and also, it must not be forgotten that this condition is frequently associated with marasmus, or may follow any of the infectious diseases of infancy or childhood.

The presence upon their favorite sites of one or several painful conical elevations that suppurate and express a "core" will establish the *diagnosis* of furuncle. A boil is frequently

mistaken for a "carbuncle." The latter condition, however, is serious. Chill and elevated temperature are early symptoms. The skin is hard, and is not freely movable; local infiltration is pronounced. Several pustules appear which indicate sites of resulting necrosis. Fortunately carbuncle is rarely seen in infancy and childhood.

**Treatment.**—Without a doubt the most effective treatment is incision, followed by the application of a wet compress of a weak solution of *Bichloride of Mercury*. Internal medication is always indicated.

*Hepar sulph. calc.* is an ideal drug, not only for this condition, but also for all pustular dermatoses. *Sulphur* is frequently given with brilliant results. *Silica* is especially indicated in those cases which tend toward furunculosis. The early administration of *Belladonna*, and occasionally of *Aconite*, will surely minimize pain, and may abort an attack. It must be remembered that poor hygiene, uncleanly and unsanitary surroundings and improper food, play a most important rôle in the production of pustular diseases; where practicable, these conditions must be corrected.

#### IMPETIGO.

Although very few cases of non-contagious impetigo have been observed, yet because of the claims made by Duhring ("Cutaneous Medicine"), Stellwagon ("A System of Genito-Urinary Diseases, Syphilology and Dermatology"), and Hardaway ("An American Text-Book of Genito-Urinary Diseases, Syphilis and Diseases of the Skin"), dermatologists accept a simple non-contagious type of impetigo.

**Definition.**—Impetigo is an acute non-contagious dermatitis, characterized by the formation of pustules.

**Symptomatology.**—This condition is recognized by the presence, chiefly upon the face and extremities, of a varying number of pustules about the size of a pea. These lesions, which appear at times during the course of the disease, are discrete, each being surrounded by an inflammatory base.

They are well distended with a straw-colored fluid, which ends in crust-formation without rupture or umbilication. Scars never result. Itching and burning may be an announcing feature.

**Etiology.**—Very nearly all cases occur during infancy and early childhood. It is a local infection, for which the staphylococcus is responsible.

**Diagnosis.**—Impetigo simplex resembles closely impetigo contagiosa, and in atypical cases a differential diagnosis is beset with difficulties. The contagious variety is recognized by lesions of a vesico-pustular character. The lesions of impetigo simplex are invariably pustules. Umbilication is seen only in the contagious type.

**Prognosis.**—This disease is an acute, self-limited process.

**Treatment.**—Removal of the cause by proper observance of cleanliness will produce a prompt cure.

#### IMPETIGO CONTAGIOSA.

**Definition.**—Impetigo contagiosa is an acute contagious dermatitis, characterized by the formation of superficial, circular or oval vesico-pustules or blebs, which rapidly form yellowish crusts.

**Symptomatology.**—Except in isolated cases, occurring in infants, no constitutional symptoms precede or accompany an attack. When present, however, they give rise to submaxillary and pre-aural adenopathy, together with moderate fever. The lesions are usually seen upon the face and hands. When the fingers become involved the lesions are situated about the tissues surrounding the nails. Exceptionally, lesions are found on the trunks and extremities.

The lesions at first are minute vesicles, later increasing in diameter, becoming vesico-pustules. Their contents are sero-purulent. Desiccation rapidly occurs, leaving brownish spots, which soon disappear. The attack lasts about a week, fresh crops appearing daily. Occasionally lesions rupture and coalesce, giving a honeycomb appearance to the group; under such conditions itching is a prominent feature.

**Etiology.**—Impetigo contagiosa is due to filth, and is rarely seen except in dispensary practice or among those who are improperly cared for. The disease is very contagious. Adults, however, are rarely attacked. Authorities trace the disease to the presence of the staphylococcus aureus et albus.

**Diagnosis.**—Impetigo contagiosa may be mistaken for impetigo simplex, varicella, the pustular type of eczema, ecthyma, and pemphigus. The features which distinguish the simple and contagious types have been mentioned while discussing the former variety.

From *varicella* it may be differentiated by the presence of lesions of a vesicular or bullous character, which appear in crops, and which in some instances leave cicatrices. Varicella is occasionally accompanied with grave constitutional symptoms. *Pustular eczema* may suggest impetigo simplex, although an eczema invariably produces more infiltration and more subjective symptoms. In eczema the lesions, although pustular, are deeper, and surrounded by an inflammatory areola. The lesions are found upon the legs, regions rarely attacked in impetigo contagiosa. *Ecthyma* is a disease of adult life.

*Pemphigus* is rarely met with in infants and children. The lesions are blebs. Constitutional symptoms are present.

**Prognosis.**—Under appropriate treatment a rapid recovery may be looked for.

**Treatment.**—Warm baths should be given morning and evening. Crusts, if adherent, may be removed by soaking with olive oil. A mild antiseptic local application, such as the ammoniated mercury, ten grains to the ounce of vaseline, will cure the majority of cases promptly. *Hepar sulph. calc.* may be required to help eradicate the condition.

#### URTICARIA ; HIVES.

Urticaria is an inflammatory cutaneous affection characterized by the appearance of evanescent pinkish elevations (wheals) which are accompanied by considerable itching and other sensory disturbances.

**Symptomatology.**—The lesion of urticaria is a wheal. This begins as a red, slightly elevated spot which enlarges, the centre becoming paler in color. In shape it is round or oval, frequently changing its size and locality, appearing from time to time upon different portions of the body. The lesions are particularly evanescent; they may last a few hours or but a few minutes, leaving behind no trace of their former presence. Rarely they persist for days; occasionally they coalesce and attain considerable dimensions. Their favorite seats are the extremities and buttocks, although they may appear on any portion of the skin or mucous membrane. Their outbreak is invariably attended with intolerable burning and itching, and a slight degree of fever. An attack may be acute or chronic. The acute attack is usually attended with gastric derangement, headache and slight fever. The eruption appears and disappears quickly, leaving no trace save a few scratch-marks, resulting from the itching. The chronic type may last for weeks or months.

**Urticaria papulosa** is a skin affection very common in childhood. It is characterized by the appearance of small, discrete, round papules—often beginning as a wheal, but persisting as an itchy eruption. They are mostly confined to the extremities. Another form frequent in childhood is *urticaria pigmentosa*, in which a pigmented spot persists after the disappearance of the wheal.

**Etiology.**—Hives arise from causes that are both internal and external. Certain seasons are, in a measure, responsible for their outbreak; they are especially apt to appear in the spring and fall. Occasionally they accompany attacks of eczema and pemphigus. The majority of cases occurring in children may be traced directly to some gastro-intestinal derangement. Constipation, diarrhœa, worms, and acute or chronic indigestion may occasionally be responsible. Improper clothing, low or high temperature, and the bites or sting of insects may be exciting causes.

**Diagnosis.**—The character of the wheals, their evanescence

and their arrangement, associated with intolerable itching and tingling, are sufficient to establish the diagnosis. The eruption may be mistaken for *eczema papulosum* and *pemphigus*. Eczema papulosum, however, presents lesions of a papular type, which persist for a longer period. In pemphigus the lesions are bullæ. Moreover, pemphigus is a rare disease in childhood. There is usually marked constitutional disturbance in pemphigus.

**Prognosis.**—The prognosis is favorable in the active variety. Removal of the exciting cause, usually a gastrointestinal derangement, will effect a cure. In the chronic form the tendency to relapses must always be borne in mind.

**Treatment.**—The treatment of urticaria is simple. Articles of diet which disagree must be interdicted. Constipation or diarrhœa, if present, must be corrected. During an attack the diet must be of the plainest kind. Locally it may become necessary to allay itching by applying a weak solution of *carbolic acid*, one-half of a drachm to eight ounces of water, or hot water to which has been added a little vinegar.

*Aconite* may be administered early in an attack to control the fever, thirst and restlessness.

*Urtica urens* is indicated when itching, burning and tingling are prominent symptoms. It is indeed almost a specific.

*Antimonium crudum*, *Arsenicum*, *Nux. vomica* and *Pulsatilla* are of service in cases arising from gastric irritability.

In the chronic form *Hepar sulph. c.* may be looked upon as specific.

#### VEGETABLE PARASITIC DISEASES; TINEA.

The term *tinea* embraces the vegetable parasitic diseases of the skin. Those due to the trichophyton fungus are spoken of as *tinea trichophytina*. *Tinea trichophytina* affects the scalp and body.

## TINEA TONSURANS.

**Synonyms.**—Trichophytosis tonsurans, ringworm of the scalp.

**Definition.**—Tinea tonsurans is a highly contagious vegetable parasitic disease of the scalp, characterized by the presence of one or several bald spots, covered with scales and containing short, broken-off hairs.

**Symptomatology.** — Following a period of incubation, variously estimated at from three to five days, erythematous areas about the size of a twenty-five-cent-piece appear. They are covered with grayish scales, and are accompanied by slight itching; they enlarge peripherally and may coalesce. The hairs of these parts become lustreless and break off. In some cases the scalp is entirely denuded, making a complete bald spot. Occasionally vesicles and pustules form, and a certain amount of suppuration results. Resolution may take place in one area, while the disease is active in another. The general health is rarely affected.

**Etiology.**—Tinea tonsurans is due to the presence and growth of the trichophyton fungus. It is highly contagious, being transmissible to the lower animals, from whom it may be contracted. It is often endemic in asylums and hospitals, or where a number of children are congregated.

**Pathology.**—As a rule, only the superficial parts of the epidermis and hair are attacked in children. Microscopically mycelia and spores are seen. The hairs become brittle, but, as a rule, baldness is not permanent. The hairs usually return to their normal state.

**Diagnosis.**—Ringworm of the scalp may be mistaken for *alopecia areata* and *squamous eczema*.

*Alopecia areata.*—Baldness in alopecia areata is complete. The condition develops quickly. It may be associated with ringworm of the scalp.

**Prognosis.**—Isolated cases, if seen early and subjected to proper treatment, are curable within a few weeks. An epi-



**demio** occurring where a number of children dwell together is hard to eradicate. In the majority of cases a few months will be required to efface the disease, and it must be remembered that relapses are common.

**Treatment.**—The treatment of ringworm of the scalp is difficult and tedious. Internal remedies will be required to prevent suppuration, in which event *Hepar* is indicated, or, where anæmia or scrofula exists, *Arsenicum*, *Thuja*, *Mercurius* and *Sulphur* may be advantageously employed. The best results in all cases are obtained from the application of parasiticides. It first becomes necessary to place the scalp in a condition suitable to receive local treatment. The hair about the patch and for some space surrounding it should be cut and the scalp closely shaven. The short hairs should be removed by means of suitable forceps. Scales and crusts, if present, are removed by scrubbing vigorously with a solution of green soap. Where the patches are extensive, it is necessary to shave the entire scalp. Depilation of the diseased hairs is tedious and often unsatisfactory. As a rule the hair is brittle and breaks off, not coming out entirely. The process, however, should be practiced daily. Locally the best application is *Bichloride of Mercury*, one to one thousand; it should be discontinued if it excites active inflammation. *Carbolic acid*, one drachm to one pint of water, is frequently efficacious. Among other agents are *Sulphur ointment*, a five per cent. ointment of the *Oleate of Mercury*, and equal parts of the *Oil of Cade* and *Olive oil*. After an apparent cure, the scalp should be treated every other day, to prevent the possibility of a relapse.

#### TINEA CIRCINATA; RINGWORM.

*Tinea circinata* is a highly contagious vegetable parasitic disease of the skin, caused by the *trichophyton* fungus. It is characterized by the presence of several patches of varying size and character, occurring upon any part of the body surface.

**Symptomatology.**—Ringworm of the scalp and ringworm of the body are often found co-existing. Minute, irregular-shaped spots of a reddish-brown color indicate the commencement of ringworm of the body. Later a distinct circular patch is seen, which heals in the centre and spreads peripherally. Around the margin of each patch small papules and papulo-vesicles are seen. Scaling is a distinct feature. The typical ringworm is usually about the size of a dime, and it stands out prominently from the surrounding skin. In some instances the rings join together. Any part of the body may become affected, although the face and hands are most frequently attacked. Next to these localities, the axillary and inguinal folds are involved.

**Etiology.**—*Tinea circinata* is due solely to the presence of the trichophyton fungus. The disease is highly contagious. Adults are often attacked. It is, however, more common in childhood.

**Diagnosis.**—*Tinea circinata* may be mistaken for *seborrhœa* and *eczema squamosum*. In seborrhœa the scales are greasy and the fungus is absent. Should any doubt exist as to the diagnosis, a microscopical examination will usually detect the fungus.

**Prognosis.**—An acute attack is quickly curable, but in the anæmic and poorly nourished it may be quite rebellious to treatment.

**Treatment.**—The fungus can be destroyed by scrubbing the lesions every morning and evening with green soap and hot water, and afterwards applying a solution of *Sodium Hyposulphite* (drachm to the ounce) or painting the patch with a weak *Iodine* tincture. In obstinate cases it may be necessary to resort to a 25 per cent. aqueous solution of *Ichthyol*. Care must be observed in using *Ichthyol*, since it is likely to provoke an acute dermatitis. Internally *Hydrastis*, *Natrum muriaticum*, *Sepia* or *Graphites* may be indicated.

## ANIMAL PARASITIC DISEASES: PEDICULOSIS ; LICE.

**Definition.**—Pediculosis is a contagious animal parasitic disease, in which the body is infested with lice. These set up both primary and secondary lesions.

**Symptomatology.**—In infants and children pediculosis is, as a rule, confined to the scalp. The uncleanly are mostly attacked. These parasites attack the scalp, causing much itching and scratching ; escape of serum and purulent fluid occurs, forming crusts. The hairs become matted together ; scratch-marks, pustules, excoriations and furunculi contribute to this unsightly condition. The cervical glands become tumid and enlarged.

Occasionally an eczematous condition of the scalp accompanies *pediculus capitis*. Pediculi are found both upon the scalp and the hairs. Their nits are usually upon the hairs. The term *plica polonica* has been applied to an aggravated state of lousiness, where living and dead lice and their nits have matted the hairs together, a most offensive odor arising from the decomposing pus and crusts. Severe inroads are in some instances made upon the general health, traceable to the annoyance coincident to incessant itching and scratching.

**Diagnosis.**—The detection of pediculi and their nits, together with their resulting secondary changes, will at once establish the diagnosis.

**Treatment.**—Naturally local treatment is indicated. Kerosene oil is the best remedy with which to kill the parasites and their ova. It should be applied freely, and the scalp subsequently covered with a muslin or oiled-silk cap. On the following day the head should be shampooed with soap and water followed by the liberal application of diluted vinegar, which dissolves the nits. This procedure may have to be repeated a number of times before a cure is completed. Should eczema of the scalp be present it must receive suitable treatment. No internal remedy is indicated, except in debilitated subjects. These must be prescribed for symptomatically.

## SCABIES ; ITCH.

**Definition.**—Scabies is a contagious animal parasitic disease of the skin, which is produced by the *acarus scabiei*.

The male itch-mite rarely burrows beneath the epidermis. The female, however, penetrates deeply, making minute tunnels, which serve as its habitat. The acarus selects those regions where the skin is tender, as the axillary and interdigital spaces, producing papules, vesicles, pustules, bullæ, wheals, infiltrations, furuncles and crusts.

**Pathology.**—Inflammation of the papillary layer of the skin results from the presence of the acarus. Itching, which is usually intense, is a very distressing symptom. It is particularly severe during the sleeping hours, since the female acarus is most active when the patient is protected by the warmth of the bed-coverings.

**Etiology.**—Uncleanliness invites the disease. Personal contact covering a prolonged period is also responsible. The itch-mite alone is the exciting cause.

**Diagnosis.**—The diagnosis of scabies is not attended with any difficulty. The presence of characteristic lesions, situated in the interdigital and other favorite regions, associated with marked and distressing itching, should lead one to a positive opinion. Scabies may, however, be mistaken for eczema and pediculosis.

**Eczema.**—This disease presents many, but by no means all, of the multiform lesions which accompany scabies. Itching is confined to the diseased parts. Pediculosis causes itching only of the parts attacked. Itching as a symptom of scabies is frequently referred to parts unattacked. Some confusion may exist where eczema or impetigo occurs in a subject already affected with scabies.

**Prognosis.**—A rapid recovery may be expected where an antiparasitic treatment is instituted early; otherwise, scabies may assume a somewhat intractable feature.

One of the best and least irritating remedies to destroy the

itch-mite is *Balsam of Peru*. This may either be used alone—rubbed well into the infected site after thorough scrubbing with green soap and hot water—or combined with sublimated sulphur, one drachm of each to the ounce of vaseline. The treatment should be carried out night and morning for three days, after which a complete change of clothing and bed-clothes is to be made and the child given a hot bath. The clothes should be baked before putting them into the wash.

Dermatitis, if excited, may be controlled by discontinuing the use of the ointment and instituting appropriate treatment. Occasionally the health becomes undermined by reason of the incessant itching and scratching. In such instances internal remedies are indispensable.

*Sulphur* is an ideal remedy. It is particularly indicated in scrofulous and uncleanly children and is helpful in the cure of the associated lesions.

*Arsenicum* may be administered where anæmia, prostration and marasmus complicate the disease.

*Hepar sulphur. calc.* possesses decided virtues in scabies, as well as in many other skin diseases presenting lesions of a vesico-pustular character.

## CHAPTER XV.

### DISEASES OF THE BLOOD.

The total amount of blood in the body of a child is somewhat less in proportion to the body weight than in the body of an adult. Likewise the specific gravity is lower, the average being 1052 as compared to 1055 in adults. It bears a close relationship to the amount of hæmoglobin, which is also proportionately low during infancy and early childhood. In the new-born, however, the hæmoglobin percentage is high, but thereafter it rapidly falls, ranging between 55 and 85 per cent. Under ordinary circumstances 60 per cent. may be accepted as the limit of blood poverty compatible with health.

The *red corpuscles*, or *erythrocytes*, are most numerous at birth. Even during the period of infancy they remain relatively more numerous than in childhood and in adult life. They gradually decrease from six to six and a half million per cubic millimeter at birth to four and a half to five and a half million in early childhood, and the normal standard of four and a half to five million is attained later in childhood. Fluctuations in the number of erythrocytes is, however, more common than in adults; even daily variations can be observed.

The form of the red corpuscle is variable in the new-born, and nucleated corpuscles (*normoblasts*) may be seen. The corpuscles also readily lose their hæmoglobin, forming the so-called shadows of Silbermann. Variations in form, and the occurrence of nucleated red corpuscles later in childhood, are, however, always pathological (GEISSLER and JAPHA).

The *leucocytes* are relatively more numerous than in adults. In the new-born an actual leucocytosis exists. According to Hayem there may be as many as 18,000 leucocytes to the cubic millimeter, but they fluctuate widely under slight influences, such as diet. The ratio of leucocytes to erythrocytes

in sucklings is, according to Gundobin, 1-395; in older children, 1-400 to 590. Rieder found a moderately high proportion of leucocytes at birth, with a decrease on the second to fourth day, after which the number of white cells again rose.

The various forms of leucocytes are: (*a*) *Lymphocytes*, or small mononuclear cells, which are believed to originate from the lymphoid tissue. They are about the size of a red blood corpuscle and contain a single large nucleus which almost completely fills the cell. A narrow rim of strongly basophile, homogeneous or coarsely reticular cytoplasm surrounds the nucleus. Normally (in adults) they constitute about 25 per cent. of the total, but in infants there may be 50 to 70 per cent. (GUNDOBIN). The lymphocytes are most markedly increased in lymphatic leukæmia. An actual increase is also noted in many cases of rickets, and a physiological increase occurs after feeding. Passive hyperleucocytosis—due to the mechanical washing out of certain lymphatic districts into the blood current—is observed in gastro-intestinal disturbances and in whooping-cough. A relative lymphocytosis occurs during the second and third week in typhoid fever.

(*b*) *Large mononuclear cells*, derived from the bone marrow and spleen. They are much larger than the preceding form and are not so numerous constituting about 6 per cent. of the different forms. In infancy the percentage is higher, while in the foetus they are the most numerous. The nucleus is vesicular, does not stain as deeply as that of the small leucocyte, and at times has an indented, horse-shoe appearance, believed to be a stage of *transition* to the polynuclear form. The protoplasm is faintly basophile and may show a fine reticulum. On account of their light staining they are often spoken of as "hyaline cells." These cells are increased, as a rule, in conjunction with the lymphocytes, but they are especially increased in the so-called anæmia infantum pseudo-leukæmia, and in malarial fever. In malarial infection there is not only an absolute increase in the large mononuclear cells, but also a relative increase over the small lymphocytes.



They are also increased in measles, and in syphilis, tuberculosis and in typhoid fever, when these diseases become well established. A differential count of these cells, therefore, plays an important rôle in the diagnosis of obscure febrile affections. In cases of malarial infection without much fever and without quinine history the polynuclears are diminished and the large lymphocytes much increased (KRAUSS, *Jour. Amer. Med. Ass.*, Oct. 22, 1904). This also holds good in recent cases. As some difficulty may arise in distinguishing between a large and a small lymphocyte, Krauss gives the following rule: "Class all cells the size of a polynuclear cell as small unless the protoplasmic margin is relatively large, and contains scattered neutrophile granulations, which stamps the cell as a large one."

(c) *Polynuclear leucocytes*, or *neutrophiles*. They are large leucocytes with several nuclei connected by threads, therefore they are also called "polymorphonuclear." The nucleus takes the basophile stain while the protoplasm is neutrophile and contains distinct granulations. They are the most numerous of all leucocytes, excepting in infancy, constituting from 65 per cent. to 70 per cent. under normal conditions. In the new-born the polynuclear leucocytes represent about 63 per cent. of the white corpuscles and they rise to 70 per cent. in the first forty-eight hours. After that a rapid destruction of these corpuscles takes place and they fall to about 35 per cent. They are rapidly increased in infectious diseases, acting as phagocytes. These cells form the pus cells in all active suppurative processes. In infancy the percentage of polynuclear leucocytes is much lower than in mature children, ranging between 28 and 40 per cent. (GUNDOBIN, RIEDER).

(d) *Eosinophile leucocytes* are large, round, polynuclear cells containing coarse, granular bodies which stain deeply with eosin. Their affinity for this stain gives them their name. Normally but 2 to 4 per cent. are encountered, but in leukæmia there is both a relative and an absolute increase.

(e) *Mycocytes*, or *Markzellen*, being so named from their

origin in the medullary cavity of long bones. They are never found in the blood under normal conditions. They are several times larger than a red blood corpuscle, and have a single nucleus that stains but faintly. The protoplasm contains neutrophile granulations. They are found in spleno-medullary leukæmia in conjunction with an increase of the eosinophiles, and in severe secondary anæmias.

(*f*) *Mast-cells* are variously sized leucocytes, either mono- or polynuclear, their protoplasm containing strongly basophile granules. They are found in a small proportion in normal blood, but in leukæmia, and especially in the secondary anæmias of childhood, they are considerably increased.

The necessary data for a correct diagnosis of a pathological blood condition can only be obtained by a direct examination of the blood. This resolves itself into the following steps:

1. The determination of the *percentage of hæmoglobin*. To ascertain this, the Hæmometer of v. Fleischl is the instrument usually employed. A capillary tube holding a definite quantity of blood is filled by holding its free end over a drop of blood which is allowed to ooze from a puncture of the ear lobe. The blood is diluted with water in a chamber divided into equal halves, and by artificial light transmitted from below, the colored water is compared with the gradually increasing shade in the color of a wedge of properly colored glass that is passed under the other half of the chamber, in which distilled water has been placed. As soon as the two halves are identical in shade, the scale is consulted and the percentage read off.

2. The determination of the *number of red corpuscles*. For the purpose of counting the red corpuscles the Hæmocytometer of Thoma-Ziess is used. This instrument is supplied with a pipette graduated to hold one millimeter of blood and one hundred millimeters of diluting fluid. The blood is thoroughly diluted with a solution corresponding to the blood serum in density (Gower's solution) and a drop is then placed upon a counting chamber ruled off into squares so that the

number of corpuscles in a cubic millimeter can be accurately estimated by counting off a large number of these squares.

3. The determination of the *white corpuscles* is carried out on the same principle, but, as they are less numerous, a larger pipette, giving a dilution of one to ten, is employed. A 3 per cent. solution of Acetic acid is used as a diluting fluid. This destroys the erythrocytes and renders the leucocytes more conspicuous.

4. *The differential count* of the leucocytes is conducted by making a film of blood on a microscopical slide, drying it and staining with eosinate of methylene blue (Jenner's stain). Several hundreds are then counted and classified, when the percentage of each variety of leucocytes is easily computed.

5. The microscopical appearance of a fresh drop of blood is of great importance for diagnostic purposes. The shape and size of the red corpuscle, the absence of rouleaux formation, the presence of nucleated red corpuscles, the presence of parasites (plasmodium of malaria), must all be taken into consideration.

6. The *specific gravity* is obtained by floating a drop of blood in a mixture of Chloroform and Benzol of 1050 to 1060 specific gravity. A drop of blood is allowed to fall into a test tube containing ten cubic centimeters of the fluid, and, according as it drops to the bottom or floats on the surface, Chloroform or Benzol is added. When it remains suspended in the fluid the specific gravity of the latter is taken, it corresponding to the specific gravity of the blood drop. For a full description of the instruments, technique and methods of blood study the various works on hæmatology must be consulted.

#### ANÆMIA.

The various forms of anæmia occurring during infancy and childhood are not well understood. The majority of cases are purely secondary to some constitutional disturbance or dependent upon indigestion or malassimilation. The marked

difference between the blood of the infant and that of the adult, especially in the wide range of fluctuations to which it is subject both in its chemical and morphological elements and the strong tendency to reversion to the embryonic type makes it difficult to determine just where one form of anæmia begins and another ends, or even to determine definitely the actual presence of a pathological blood-state. It is becoming more and more the conviction of hæmotologists to question whether a primary form of anæmia is ever encountered in children; and whether there be such a clinical entity as *pseudo-leukæmia infantum* of v. Jaksch—a disease in which the blood shows changes similar to those observed in leukæmia, but in which recovery may take place.

Again, the mere presence of leucocytosis is not a sufficient datum for classifying an anæmia, it being necessary to determine the proportion of the various forms of leucocytes. Neither can splenic tumor be employed as a means of differential diagnosis in anæmias, as this may be present even when there is no anæmia (GEISSLER and JAPHA). In the anæmia associated with rickets, a condition of the blood showing all of the stages of anæmia may occur, from a slight diminution of the hæmoglobin and of the red corpuscles to the occurrence of magaloblasts. There also may be splenic tumor. While such a condition is sometimes described as *anæmia splenica*, still there are no grounds for looking upon it as a primary, independent disease. If we simply remember that the child's blood is particularly susceptible to deterioration, and that a strong tendency exists for the blood to revert to a less mature histological type, we will not misinterpret the various blood changes encountered at this time of life.

*Ischæmia*, or local anæmia, a condition resulting from an interference with the circulation in a localized portion of the body is a purely mechanical condition and not to be considered here.

*Secondary, Simple, or Symptomatic Anæmia* comprises the class of cases resulting from hæmorrhage, inanition, intestinal

parasites, errors of feeding, unhygienic surroundings, rickets, nephritis, long continued suppurative processes, syphilis, tuberculosis, malaria and other forms of infections and from certain poisons such as Lead, Mercury and Arsenic. Secondary anæmia may be divided into several types, the following classification being after Morse (*Archives of Pediatrics*, 1898).

*Mild anæmia*, characterized by trifling reduction in the hæmoglobin percentage and number of erythrocytes and absence of abnormal changes in the blood elements.

*Severe anæmia* with pronounced diminution of hæmoglobin and erythrocytes, together with changes in the size and shape of the corpuscles and the presence of normoblasts, or nucleated red blood corpuscles.

*Anæmias with leucocytosis* are usually associated with more pronounced reduction in hæmoglobin and red corpuscles than anæmias without leucocytosis (DA COSTA). Normoblasts and deformities in size and shape of the erythrocytes are encountered in these cases.

#### CHLOROSIS.

Chlorosis is a form of primary anæmia which is seen most frequently in girls at the time of puberty, but it is not necessarily confined to this period of life nor to the female sex. Of the *etiology* nothing positive is known excepting that unhygienic surroundings, improper or insufficient food, lack of fresh air and sunshine, emotional disturbances and obstinate constipation are frequently intimately associated with the development of chlorosis. The heart and larger blood-vessels have been demonstrated by Virchow as under-developed in many instances.

The *symptoms* of chlorosis may make their appearance rapidly, or the disease may not be suspected for a long time until pallor and the characteristic greenish tint of the skin, on account of which it is popularly known as "green sickness," give a clue to the existing ill-health. The child complains of headache, and displays an aversion to mental or

physical exertion of any kind. The latter results in dyspnoea and palpitation, while the headache and languor induce indifference both to work and to play.

The appetite is poor, and in many instances becomes perverted, so that the patient craves chalk, slate-pencils, coffee-beans, etc., which are apparently enjoyed. Indigestion and constipation are troublesome symptoms, and their correction materially hastens the cure.

In young girls, menstrual derangements are inseparably associated with chlorosis. Thus, scanty menstruation or amenorrhoea are almost invariably encountered in these cases; likewise, dysmenorrhoea and leucorrhoea are common. Improvement in the chlorotic condition results in prompt improvement here.

The red corpuscles are but slightly decreased in number, but there is a pronounced deficiency of hæmoglobin, giving the individual corpuscles a noticeably pallid appearance.

Edema tends to develop about the ankle-joints, and many patients present a puffy, fat appearance, indicating a hydræmic state, with sluggish return circulation. The degree of anæmia can be roughly estimated by the appearance of the palpebral conjunctiva, the lips and the matrix of the nails, but in order to follow the progress of the case accurately we should make weekly hæmoglobin estimations with the hæmometer of v. Fleisch, or with Dare's hæmoglobinometer. A less accurate, but very simple procedure, yet at the same time a far better method than the pure guesswork of merely inspecting the mucous membranes, is afforded us in the Tallquist scale. Here it is only necessary to place a drop of blood upon a piece of absorbent paper and compare it with the scale attached to the book.

The *prognosis* of chlorosis is favorable, and it usually responds promptly to treatment, although there is liability to relapses. As it creates a tendency to tuberculosis it becomes dangerous when occurring in individuals with tuberculous antecedents.

## PROGRESSIVE PERNICIOUS ANÆMIA.

This form of primary anæmia is a rare disease, and is more seldom seen in children than in adults. Quite a sufficient number of cases, however, are on record to assign it at least a brief mention in a work upon the diseases of children. The *etiology* is obscure. Birch-Hirschfeld advances the infectious theory, owing to the presence of tissue destruction and retardation of blood-coagulation; others hold to the theory of increased hæmolysis, and again others to decreased hæmogenesis. Stengel (*Medical News*, Oct. 20, 1900) expresses the view that pernicious anæmia is undoubtedly a disease resulting from the rapid destruction of red blood corpuscles, for the compensation of which the blood-making functions prove inadequate; and, further, that the source of the hæmolytic agents is the gastro-intestinal tract.

The anæmia resulting from intestinal parasites is very difficult to distinguish from pernicious anæmia, showing the great liability for error and the difficulty with which a study of the disease is beset, as so many agencies are capable of inducing pronounced anæmia. In eighteen cases seen by Osler (*Amer. Text-Book of Practice*) there was absolutely no appreciable cause for the disease. Henoeh (*Vorlesungen* <sup>ii.</sup> *Kinderkrankh.*) saw two children in the same family die of this disease. no cause being ascertainable. Ewing thinks that any case of pronounced, progressively-increasing anæmia in which the blood contains megaloblasts and a considerable proportion of megalocytes with increased hæmoglobin, while the lymphoid marrow shows marked hyperplasia of peculiar type, should be considered one of pernicious anæmia, regardless of the immediate exciting cause. Even in the gravest secondary anæmias these changes are rare, but in early life the changes in the blood are so uncertain that their significance is difficult to determine. The frequency of pernicious anæmia in childhood, therefore, is still a question.

The symptoms are those of a gradually increasing anæmia.



Loss of flesh may be absent. Œdema and hæmorrhage may supervene. The skin assumes a characteristic lemon-yellow tint. Anorexia, vomiting and other digestive disorders accompany the condition. The patient eventually dies from exhaustion. As the name implies, the entire course is a progressive and pernicious one. The blood changes are the same as found in the adult as far as pronounced oligocythæmia and nucleation and deformities of the erythrocytes are concerned, but the blood often fails to show the high color index and the prevalence of megaloblasts and of megalocytes that are accepted as diagnostic of the disease in adults (DA COSTA).

**Treatment.**—The hygienic management of cases of anæmia is important, and the physician must study his patient carefully before determining upon the question of diet, exercise and rest. In chlorosis it is especially important to improve the condition of the bowels, and the selection of a diet to overcome constipation is a great advantage to the patient. Fruits and fresh vegetables, many of which are rich in iron (notably spinach), are very beneficial. For anæmia in general it may be said that the most nutritious and most digestible form of food is to be selected. The impoverished and watery condition of the blood diminishes the organic elements of the digestive secretions, for which reason it is often desirable to aid the digestion by the employment of digestive ferments, such as pepsin or papain, or, as Thompson (*Practical Dietetics*) recommends, to employ predigested foods, making use of pancreatin in the preparation of animal food and diastase or malt extracts for the predigestion of amylaceous food.

Milk is an ideal food in all forms of anæmia, and chlorotic subjects may drink of it freely, even between meals. Eggs are also very beneficial, being easily digested, and their yolk contains a large proportion of iron. There is some risk in using raw beef, but meat is usually not well digested by these patients unless given practically raw. A good red wine often proves most beneficial.

Where exhaustion is a prominent symptom, rest rather than exercise should be prescribed. Absolute rest in bed, with massage and liberal feeding, will accomplish more in such cases than exercise, which only adds to the exhaustion and tissue breakdown.

The following remedies are the ones most useful in the various forms of anæmia:

*Belladonna*.—In *chlorosis*, when there is violent palpitation, throbbing headache, great weariness and desire to sleep in the afternoon, debility. The symptoms of *Belladonna* are very similar to those of *Ferrum*, especially the palpitation, dyspnœa and rush of blood to the face, alternating with paleness; but there is not that intense anæmia and persistent debility, gastralgia, vomiting, amenorrhœa and anasarca indicating the latter remedy.

*Ferrum* is seldom of use elsewhere than in *chlorosis*, to which it is strictly homœopathic, as indicated by its symptomatology. Here it has gained universal reputation, and even as prescribed empirically by the old school in large doses it is frequently of great benefit, owing to its favorable action upon the intestinal tract, by uniting with the hydrogen sulphide gas in the intestines. In this way the assimilation of the organic iron compounds present in the food is permitted, an impossibility in the presence of free hydrogen sulphide gas. No doubt such remedies as *Pulsatilla*, *Nux vom.* and *Spigelia* owe their prominent usefulness in chlorosis to their influence upon the alimentary tract, and when they are indicated the use of *Iron* is not always necessary for the cure, as sufficient iron should be absorbed from the food to supply the blood with all it requires. Iron is no doubt a specific food in chlorosis, for in this form of anæmia the erythrocytes are not diminished in great number, the chief disturbance being the reduction in hæmoglobin and the consequent low color index. Iron supplies the necessary element, and thus the hæmoglobin is restored. It is different with other forms of anæmia, in which the blood elements themselves are deficient. As iron

does not act upon the hæmatopoietic organs it cannot be expected to benefit such cases, and here we must look to remedies like *Arsenic* and *Mercury* for results. Many preparations of iron are in vogue, each form having its ardent advocates. *Ferrum redactum* in the first decimal trituration is one of the most reliable preparations; the *Oxalate of Iron* finds great favor with many of the British homœopathists in chlorosis. The *Citrate of Iron and Strychnia*, second decimal and third decimal trituration, is a preparation which stands highly recommended for cases in which debility is a prominent symptom.

*Graphites*.—*Chlorosis*, tendency to obesity, sluggish circulation and anæmia, with general coldness; delayed or scanty menses, obstinate constipation; sad, tearful disposition.

*Natrum mur.*—*Chlorosis*, obstinate cases, fluttering of the heart, craving for salt.

*Nux vom.*—*Chlorosis*, gastric derangements, constipation, irritability, prostration; languid, especially morning on rising from bed; perverted appetite.

*Pulsatilla*.—*Chlorosis*; great weakness and sluggishness of the circulation, manifesting itself as chilliness; coldness and paleness of face, relief in open air. Anorexia, nausea, palpitation of heart and dyspnœa, sharp pains about heart (compare also *Spigelia* and *Cactus*, both of which are indicated by their cardiac symptoms), amenorrhœa, leucorrhœa; sad, tearful disposition. *Cyclamen* is similar to *Pulsatilla*, but this remedy has aggravation of symptoms in the fresh air, due to great sensitiveness to cold.

Other important remedies in chlorosis are *Calc. c.*, *Helonias*, *Sepia* and *Sulphur*.

**Symptomatic anæmia** finds in *China* a most valuable drug. *China* is indicated after hæmorrhages, chronic diarrhœa, long-continued suppuration, and in all mild forms of idiopathic anæmia as a "tonic," given in doses of two to three drops of the tincture, three to four times daily.

*Arsenicum* corresponds more closely to the pernicious forms

of anæmia than any other remedy, and is also indicated in the anæmia of malaria and of Bright's disease. Its indications are excessive debility, œdema of the ankles and eyelids, cardiac weakness and dyspnœa, gastric irritability. It may also be required in severe cases of chlorosis.

*Phosphoric acid* and *Silicea* are useful in the anæmia of debilitating diseases, such as typhoid fever, following well after *China*.

*Mercurius* is specific in the anæmia of syphilis.

*Kali carb.* corresponds to a vitiated state of the **blood plasma**. Farrington refers to its ability to produce anæmia, and recommends it for the blood poverty after severe or protracted diseases. The following symptoms are recorded in Hering's *Condensed Materia Medica*: "Vertigo, congestion to head with throbbing and humming. Swelling like a bag between upper eyelids and eyebrows. Palpitation in spells, taking his breath; stitches about heart; weak, irregular pulse. Arms go to sleep. Swelling of feet to ankles. Anæmia, with great debility; skin milk-white; muscles weakened, especially the heart." Our claims for the value of this remedy in anæmia have been substantiated lately by old school therapeutics. Denstedt and Rumpf (*Therapeutische Monatshefte*, March, 1901) demonstrated that in pernicious anæmia the blood gave a high percentage of water and sodium chlorid and a great reduction in the percentage of iron and potash. Accordingly, *Potash salts* were administered in several such cases, both by mouth and infusion, with marked improvement. It seems that the death of the corpuscles depends upon the abstraction of its potash, and *Potash*, therefore, has the same specific relationship to degenerative changes in the corpuscles that *Iron* has to hæmoglobin poverty in the corpuscles (chlorosis) and *Arsenic* to the making of new blood elements, hæmatosis.

LEUKÆMIA; PSEUDO-LEUKÆMIA; SPLENIC ANÆMIA;  
HODGKIN'S DISEASE.

The varieties of anæmia described under the above titles present as their most characteristic features permanent leucocytosis and splenic enlargement.

Their differentiation presents many points of difficulty, which can only be definitely settled by careful hæmatological examinations. Aside from the pseudo-leukæmic anæmia of infants, they are seldom encountered during childhood. They all present an unfavorable prognosis.

**Leukæmia** may affect persons of all ages, but is rare during childhood. Mossa has collected a series of twenty-seven cases in children, but he admits that a large number of these were undoubtedly not cases of true leukæmia. Da Costa collected ten cases, in all of which a differential leucocyte count is recorded, confirming the diagnosis.

The symptoms are anæmia, pronounced pallor, distended abdomen, with enlargement of the spleen, and tenderness. The lymphatic glands may be principally involved, as in the *lymphatic variety*, or the spleen and marrow, in the *spleno-medullary variety*. In the *lymphatic variety* the lymphocytes are markedly increased, sometimes the large, at other times the small mononuclear cells predominating. The polynuclear cells are relatively decreased. In a case coming under my notice the polynuclear cells had almost entirely disappeared from the blood, the blood-count giving five thousand leucocytes, mostly lymphocytes. The erythrocytes are diminished and a few normoblasts may be present. In the *spleno-medullary variety* there is a relatively small increase in the lymphocytes, but myelocytes are found in abundance in conjunction with an increase in the eosinophile cells.

The disease assumes a progressively downward course, usually terminating in general œdema, hæmorrhages and exhaustion. At times it is febrile, simulating an infectious disease, and runs an acute course.

**Hodgkin's disease** presents enlargement of various groups of the lymphatics; enlargement of the spleen and liver; fever of an intermittent type, and progressive anæmia and leucocytosis; but the latter never attains to the degree found in leukæmia. The cervical and axillary glands, or those situated near by, are usually the ones first affected, other groups eventually becoming implicated. They do not, however, tend to break down, this being a strong point of differentiation between Hodgkin's disease and *tuberculous adenitis*. The course is chronic, and although the child may live for a long time, still it ultimately succumbs.

**Pseudo-leukæmia**, or *Anæmia Infantum Pseudo-leukæmica* (v. JAKSCH), is a disease of childhood, usually seen before the second year. The *etiology* is obscure. It was first described by v. Jaksch, its characteristics being: Occurrence in infancy; oligocythæmia and oligochromæmia; permanent leucocytosis; marked splenic enlargement, and at times lymphatic enlargement. The liver is but slightly enlarged, a clinical distinction between this disease and leukæmia. The prognosis is more favorable than in the latter disease, but many cases prove fatal nevertheless. The term **splenic anæmia** has been applied to a class of cases similar in all respects with the above, excepting in that leucocytosis is absent.

The development of pseudo-leukæmia is one of progressive pallor, failure in general health, digestive disturbances, and at times slight pyrexia. The anæmia is very noticeable, and palpation reveals an enlarged spleen. No doubt many cases described as pseudo-leukæmia are in their true nature really nothing more than aggravated types of rickets, for anæmia, digestive derangements and enlarged spleen are all found in well-developed rickets. As has been observed above, the present tendency is to doubt the actual existence of this form of anæmia as a separate disease. The possibility of a malarial and syphilitic influence must also be excluded. The *course* is the same as in the other leucocytoses, the characteristics being chronicity and lethal termination, although the chances

for recovery are greater in pseudo-leukæmia than in Hodgkin's disease or in true leukæmia.

**Treatment.**—Homœopathic literature on these affections is meagre. Of our writers, Gilchrist enters most extensively into the subject in an article upon "Leucocythæmia" (*Arndt's System of Medicine*) [leukæmia], in which he also reports a case of Dr. Gaylord's represented as leukæmia, which, however, should be classed as a case of anæmia infantum pseudo-leukæmica. The patient was an infant of six months, anæmic from birth, living in a malarial district. There was leucocytosis and splenic enlargement. *China* 2x and an occasional dose of *Ferrum* resulted in a cure. Dr. Broadbent (*Hom. Review*, vol. xxi) recommends *Phosphorus* as the most appropriate remedy in leukæmia, and v. Grauvogl (*Lehrbuch der Homœopathie*) considered *Natrum sulph.* and *Thuja* as most prominently indicated, for he considered these disturbances as a form of "sycosis." Gilchrist believes *China* and *Phosphorus* to be the most closely related remedies to the disease. The old school ties to *Arsenic* in conjunction with *Iron* and *Codliver Oil* in leukæmia, pseudo-leukæmia and Hodgkin's disease, although they claim no positive results from this form of treatment. Koplik has used *Ichthyol* with some success in leukæmia. Owing to its strong homœopathic relationship to rickets I should look upon *Phosphorus* as the most appropriate remedy in the so-called "splenic" and "pseudo-leukæmic anæmias." *Ficric acid* is also recommended on account of its supposed homœopathicity to leucocytosis.

#### HÆMOPHILIA.

The subjects of hæmophilia are commonly known as "bleeders," from the tendency to profuse and often uncontrollable hæmorrhages which this form of constitution presents. The disease is hereditary, and the mode of transmission is a clear demonstration of atavism through the



female, as hæmophilia rarely occurs in females, being transmitted by the daughters of bleeders to their male offspring.

The *pathology* of hæmophilia is not understood. In some instances it would seem to depend upon an abnormality in the walls of the small blood-vessels, and in others upon a delayed coagulation of the blood. The peculiarity which some cases present of only bleeding excessively in certain localities would favor the first-mentioned explanation.

The diathesis usually develops early in childhood, by the end of the first dentition period, when an accidental cut or injury first attracts attention to this tendency. Beside the danger of hæmorrhage from a traumatism or an operation, there is even as great a one from spontaneous hæmorrhage, such as epistaxis, hæmatemesis, hæmoptysis, hæmorrhage from the mouth, intestines, urethra, etc. Injuries without destruction of continuity of the skin are followed by profuse bloody effusions into the subcutaneous structures.

The hæmorrhagic diathesis cannot be recognized until a hæmorrhage has taken place, and the subjects are usually healthy-looking, apparently robust individuals, characteristically supposed to have blonde or reddish hair, blue eyes, and a fair, transparent skin. There is a strong tendency to joint-affections of a painful type, which may resemble rheumatism of the larger joints closely. When a single large joint is involved in a child it is frequently mistaken for a tuberculous lesion. A hæmorrhage may be preceded by an attack of arthritis or circulatory disturbances, such as oppression, palpitation, and rush of blood to the head.

The *prognosis* is always grave, one-half of the cases dying before the seventh year. As there is a tendency to outgrow the condition, the prognosis becomes more favorable with advancing years. There seems to be no untoward effect upon the functions of menstruation and parturition in female bleeders; another argument in favor of the origin of the disease is the vascular system, probably an inherited inefficiency in the endothelium of the capillaries distributed over certain areas.

**Treatment.**—Powers (*Surgical Diseases of Children*) advises against the use of styptics in hæmophilia, as they are always useless. A case has come under my notice in which the thermo-cautery had to be resorted to after the extraction of a tooth. The application of fresh blood to the wound has acted successfully (BIEUDWALD). The inhalation of carbonic acid gas (WRIGHT, *British Med. Jour.*, 1894) has a decided influence over the epistaxis, which may also require plugging of the nares. Supra-renal extract is a most powerful styptic and less objectionable than tannin or perchloride of iron. Gelatin is highly recommended by some surgeons.

As a constitutional remedy *Phosphorus* corresponds most closely to the condition. The remedies which have won favor in the control of hæmorrhages of various types, such as *Erigeron*, *Crocus*, *Hamamelis*, *Secale*, *Carbo veg.*, *China* and *Bell.*, may prove of use in special cases.

#### PURPURA.

Purpura, or *morbis maculosus*, includes a variety of affections characterized by the development of reddish macules of varying size, indicating extravasation of blood into the skin.

It occurs *symptomatically* after the administration of certain medicinal substances (*Iodide of Potash*, *Quinine*, *Belladonna*); in the course of certain of the infectious fevers, notably in septicæmia, cerebro-spinal meningitis, small-pox and sometimes in measles; and from cachetic, mechanical and neurotic influences. *Primarily* it is observed in the following clinical forms: *Purpura simplex*, *purpura rheumatica*, *Henoch's purpura*, and *purpura hæmorrhagica*.

**Purpura simplex** is characterized by the appearance of crops of purpuric spots, mainly upon the legs, which may be accompanied by slight fever, articular pains and diarrhœa. The spots are bright red in color, do not disappear upon pressure, and gradually fade to a purplish and later to a greenish or dirty-yellow shade. as is the course pursued by all pur-

puric spots. The duration is short, seldom exceeding ten days. A rheumatic history is often present.

**Purpura rheumatica**, or **peliosis rheumatica** (SCHÖNLEIN), as the name implies, bears a strong relationship to rheumatism. The purpuric rash develops in conjunction with multiple arthritis. The onset is usually that of an atypical rheumatic fever: lassitude, fever, sore throat, articular pains, and in the course of a few days the rash appears, which may be associated with urticaria. It is more common in adults than in children.

**Henoch's purpura** is, according to his own description, a complicated clinical picture, in which vomiting, intestinal hæmorrhage and colic are associated with the purpura and articular swellings found in the above-described variety. (*Vorlesungen ü. Kinderkrankh.*). The prognosis of this variety is usually favorable, Henoch reporting six cases, with recovery in all, and Osler eleven cases, with three deaths (*Amer. Jour. of Med. Sciences*, Dec., 1895). The diagnosis is often beset with difficulty, especially when there are no external signs of purpura. The symptoms may be entirely abdominal and I saw a boy operated upon for appendicitis in whom the appendix was found to be normal, but the mesentery bled freely from the slightest touch. A few days later he developed a purpuric rash. Two other cases that came under my notice had been treated for gall-stone colic for a long time before the true nature of the condition was suspected.

**Purpura hæmorrhagica** (*morbis maculosus Werlhofii*) differs from the above forms of purpura in the absence of distinct rheumatic manifestations, and in the predominance of the hæmorrhagic disposition. It most frequently develops in young, delicate girls, the onset of hæmorrhages being preceded by several days of languor, headache, loss of appetite, and even moderate fever. The cases that I have seen in infants proved fatal. The worst case I ever saw, a boy three years old, was presented by Prof. Bigler at one of his clinics. One of the ears was destroyed and a leg had to be amputated. The child eventually recovered.

The spots may extend over the entire body, their size varying from that of a pin-head to fairly large blotches. The macules are often interspersed with vesicles, produced by circumscribed hæmorrhages into the rete Malpighii. The cutaneous hæmorrhages are followed by bleeding from the mucous membranes and internal organs, particularly from the kidneys. The duration is from ten days to two weeks in favorable cases. Death may result from gradual exhaustion, or from a sudden extensive hæmorrhage or a cerebral hæmorrhage.

**Purpura fulminans** is a variety of purpura hæmorrhagica occasionally seen in children. It is characterized by its rapidly-developing cutaneous hæmorrhages, which may prove fatal before other hæmorrhages have had time to manifest themselves. It offers the worst prognosis of any form of purpura.

**Treatment.**—In cases of simple purpura and the rheumatic forms in general, the best results will be obtained by prescribing for the underlying constitutional condition. Such remedies as *Bryonia*, *Ledum*, *Arnica*, *Hamamelis*, *Rhus tox.* and *Sulphur* will suggest themselves.

In the hæmorrhagic form a different line of remedies is indicated. *Crotalus*, *Lachesis*, *Kali hydrojodicum*, *Phosphorus*, *Rhus venenata*, *Secale*, *Sulphuric acid* and *Ledum* are to be consulted as most homœopathic to this condition.

## CHAPTER XVI.

### DISEASES OF THE NERVOUS SYSTEM.

The investigation of the nervous system in children presents many difficulties. Not only is the child unable to express its sufferings or describe its condition with any degree of accuracy, but we are confronted also with the task of differentiating conditions arising out of purely reflex and transient (toxic) causes from those of a more serious and permanent nature.

In many forms of disease the morbid condition becomes at once apparent, and we are enabled to study the pathological processes involved by direct observation. Cutaneous eruptions, iritis, a broken bone—these are conditions which we can observe in a purely objective manner and at once recognize. In nervous diseases, however, the lesion is hidden, and a diagnosis can be made only by determining the nature and seat of this lesion by a process of deduction. Thus in our examination we may find paralysis, atrophy, contracture, tremor or convulsion, and by studying these symptoms in conjunction with the history and associated symptoms, we are furnished with the data for a diagnosis.

This necessitates a careful study of every symptom in the case and a full recognition of its clinical significance, for in no other way can we reach a decision as to the character and location of the pathological process under consideration.

Certain physiological peculiarities of the nervous system in infancy must be recalled in order to understand the meaning of some of its disturbances. Thus, the rapid growth and immaturity of the brain predispose it to certain functional and inflammatory disturbances rare at a later period. The inhibitory centres not being fully developed, and functioning only imperfectly, slight reflex irritations which an older child would disregard are translated into motor or vasomotor dis-

charges of more or less gravity. Again, slight organic lesions, by reason of the secondary degenerative changes following them and the interference with the growth and development of adjacent parts at a time when the brain should be uniformly and rapidly developing, leave behind them the most serious and oftentimes obscure consequences.

An objective examination of the child may reveal abnormalities in the development of the body as a whole or only in certain parts. Thus, the head may be too large or too small, irregular in outline or abnormal in shape. Likewise, abnormalities in the muscular system are readily noted—atrophy or hypertrophy; paresis or paralysis of certain muscle-groups, with resulting deformity, or peculiarity of the gait; incoördination of motion, tremor, localized or general convulsions, and choreiform movements. The state of the pupils and of the reflexes, and the presence of anæsthesia or hyperæsthesia, are also of significance.

The *knee-jerk* is best elicited by having the child in the dorsal position, with the heel resting upon the examiner's hand (Fig. 13). It is exaggerated in lesions affecting the upper neurons, *i. e.*, cerebral lesions. In lesions of the lower neurons, *i. e.*, spinal cord and spinal nerves, it is diminished or abolished (poliomyelitis, diphtheritic paralysis).

*Kernig's sign* is found in meningitis (about 85 per cent. of cases), and at times in cerebellar hæmorrhage and in lesions at the base of the brain. It is a phenomenon of hypertonia of the muscles. This condition was originally described as an inability to extend the leg upon the thigh when in the sitting posture, owing to tonic spasm of the hamstring muscles. When the dorsal decubitus is assumed, the leg can be straightened out, but if the thigh is now fixed upon the abdomen it again becomes impossible to straighten out the leg and a spasmodic resistance is noted in the contracted muscles. Fig. 46 shows the most satisfactory manner of determining Kernig's sign. *Babinski's sign* is an alteration in the type of response of the plantar reflex, there being hyperextension of the great

toe instead of flexion. It indicates a disturbance in the pyramidal tracts.

It must be remembered that the majority of brain lesions during infancy and early childhood are either cortical or basilar. Hæmorrhage into the internal capsule is rare, but occurs at times in syphilitic subjects.

The function of the cranial nerves is determined in the manner employed for adults, as far as that can be carried out.

*Motor paralysis* is detected by observing whether or not the child is able to move its extremities. Inability to walk may be due either to paralysis or to rickets (rachitic pseudo-paral-

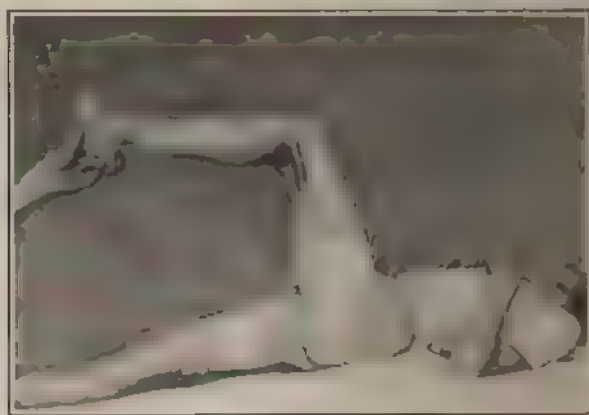


FIG. 40. METHOD OF ELICITING KERNIG'S SIGN

ysis). In the latter case the child can move the legs, as tickling the sole of the foot will prove, but it is unable to stand or to walk. *Spasticity*, or "lead-pipe rigidity," is found in cerebral palsies, usually in association with impaired mentality.

The *mental development* is difficult to gauge in infancy. The early signs of subnormal intellectual capacity, or idiocy, are inability to support the head, amaurosis (amaurotic family idiocy), crying without cause, backwardness in grasping, inability to nurse properly. Later, the time when the child



begins to walk and talk, affords important data, as well as the habits and disposition. Normally, a child should walk by the eighteenth month and begin to talk shortly after. According to West, a backward child would be normal were it of a younger age, while an idiot is abnormal for any age.

*Reaction of degeneration.* By the "reaction of degeneration" is meant that series of phenomena which takes place in a muscle supplied with a motor nerve whose spinal ganglion cell has been destroyed, or, in fact, whose lower neuron has been affected at any point in its course. The reaction is distinctive and differs so markedly from the reaction obtained by the galvanic current in a normal muscle that it serves as a ready and accurate diagnostic sign.

Briefly stated, the muscle loses its irritability to the faradic current, while the contraction with the galvanic current becomes slow and tetanoid in character, the main change, however, being that it first responds to the anodal closure with a gradually increasing current instead of to the cathode, as occurs normally. The reaction of degeneration is found typically in poliomyelitis anterior. It also occurs in progressive muscular atrophy and in multiple neuritis.

By this method of examination the location of a lesion is ascertained, but its real character can at times only be defined by extending our researches back to the child's previous history and to the family history. Knowing the prominent rôle played by such constitutional diseases as rickets, tuberculosis, syphilis and rheumatism in the etiology of nervous diseases in children, and the possibility of an identical clinical manifestation resulting, *e. g.*, from a tuberculous lesion in one case and from a syphilitic one in another, the importance of this mode of research becomes at once apparent.

#### INSANITY.

Insanity as a primary affection is rare during early childhood, but at the period of puberty it readily occurs in the offspring of neuropathic parents. Traumatism, inflammatory

affections of the brain and other nervous diseases, such as epilepsy and hysteria, are causes next in importance to heredity. In young children a moral perversion may show itself as a result of deficient intellect, this, probably in all cases, being due to some actual physical defect in the child's brain or body.

Children in whom the tendency to mental derangement is present are generally dragged, so to speak, into their insanity through mental overwork, grief, shame, excessive emotional excitement, maltreatment, masturbation and similar factors. In other words, as soon as a severe strain is brought upon the nervous system it gives way, and the child's mental instability becomes at once apparent.

While insanity in children may assume a variety of phases, its manifestations cannot extend beyond the simplicity of the child's mentality. Juvenile insanity, therefore resembles the insanity of adults in every respect, excepting in that it is limited in its development. Again, certain forms of insanity observed in the adult are scarcely, if ever, observed during childhood.

*Moral insanity* usually shows itself early, as it depends upon a deficiency in the entire intellectual sphere. The moral sense itself is deficient, and the general weakening of the intellectual faculties hinders the control of the immoral outbursts that is exercised to a greater or lesser extent by those of immoral tendency, but with good intelligence. If the immoral tendencies are sexual and pronounced already in childhood, Bayley, among others, recommends radical measures, viz., unsexing the patient. Some writers of large experience with this class of cases believe that the highest form of mental training offers a good chance of cure by developing inhibitory control.

*Mania* may occur as a primary affection or be a symptomatic accompaniment of epilepsy, hysteria or chorea. It may develop after typhoid fever.

*Epileptic insanity* manifests itself as attacks of mania, with strong tendency to dementia or mental enfeeblement.

*Hysterical insanity* presents that form of mental instability in which the emotional faculties are unduly and unrestrainedly exercised, while there is a notable lack of will-power, which may culminate in outbursts of violence, accompanied by alternate sobbing and laughing, or it may appear as the graver psychoses represented by hystero-epilepsy and catalepsy.

*Melancholia* is seldom seen before the eighth year, but it is a common form of the insanities among children. The same course is pursued as in adult cases. Both mental and physical depression, with self-persecution, characterize the condition. Attempts at suicide are not infrequent. The prognosis as to recovery is good, but a tendency to recurrence of insanity later in life is apt to persist.

*Periodic and circular insanity* is almost unknown in children. In periodic insanity there are successive attacks of mania or melancholia, alternating, perhaps, with lucid intervals; circular insanity is characterized by an alternation of maniacal and melancholic stages, followed by a lucid interval, with recurrence in the same or very rarely reverse order.

*Paranoia, primary delusional insanity, or progressive systematized insanity*, is a form of mental aberration of obscure and indefinite origin, gradually evolving with the growth of the individual.

In its fully developed state delusions of persecutions and grandeur are developed, existing as fixed ideas, which it is impossible to overcome or eradicate. Long before mental disease is apparent or suspected the paranoiac gives evidence of being odd or peculiar. Kraft-Ebing has observed that many cases of paranoia have shown strong evidence of mental instability in childhood. He could trace the incubation symptoms back to the fourth year. These children are imaginative and dissatisfied with their home surroundings, believing themselves less favored than their brothers and sisters. They are given to day-dreaming and one of the first delusions may be that they imagine themselves to be the children of other

parents—persons of higher social standing (*Lehrb. d. Psychiatrie*).

Exclusiveness; mental precocity in philosophical, religious and inventive directions; irritability of temper and cruelty are signs offering a sad outlook for the child's future mental state, especially when they are encountered in a family with psychopathic tendencies.

*Dementia*.—Paretic dementia is exceedingly rare during childhood; however, such conditions as epileptic insanity and masturbation insanity, which are relatively common during childhood, show a strong tendency to terminate in dementia, thereby frequently leading to its development at a much earlier period than usual.

*Masturbation insanity* is a form of mental disease in which masturbation exists as an uncontrollable condition, eventually developing a complete intellectual breakdown. In spite of the best-directed efforts the habit can seldom be brought under control, the patient gradually degenerating into a state of idiocy or dementia. As the case progresses the habit is so engrafted upon the mind that it practically becomes a mental process, and all sorts of devices are utilized to evade detection. It is not at all rare to find the habit already established long before puberty. A tendency to masturbate exists in all mental derangements in children, but never to such a pronounced degree as in this special form of disease.

**Night terrors** and *morbid fears* are temporary mental disturbances in which hallucinations of various kinds are developed in the child's imagination through fright, or through the suggestions resulting from the recital of ghost-stories and fairy-tales, or from vicious threats. The rational explanation for a large number of cases of *frightened awaking from sleep* is in my belief spasm of the glottis or some other form of respiratory obstruction coming on at night as a result of *adenoids*. As many children with adenoids present these symptoms the throat should always be examined in such cases. Another common cause of *disorders of sleep* is gastro-intestinal irrita-

tion; but in these the symptoms are reflex in character, and do not approach the nature of a psychosis, as do the above. In neurasthenia and lithæmia similar disturbances are observed. The idiopathic fears and terrors point to a highly neurotic form of constitution, and they may indeed be the forerunners of a more serious mental trouble.

#### IDIOCY AND IMBECILITY.

The term "feeble-minded" is employed to include all cases from the mere mentally backward down to the so-called imbecile or idiotic, the distinction being only one of degree. Imbecility denotes a lesser amount of mental incapacity than idiocy, which is thus defined by Ireland (*Mental Affections of Children*): "Idiocy is mental deficiency, or extreme stupidity, depending upon masturbation or disease of the nervous centres, occurring either before birth or before the evolution of the mental faculties in childhood."

In the etiology of idiocy hereditary transmission plays an important rôle; of all mental derangements it is the one most frequently propagated by descent. A neuropathic family tendency; parental imbecility or insanity; consanguine marriages; the tuberculous diathesis; drunkenness, and worry or fright of the mother during pregnancy, are all well-established causes of idiocy. The lesions responsible for idiocy are either present at the time of birth, having developed *in utero*, as in genetous idiocy, or they may develop late, as in the case of traumatic, inflammatory, epileptic and paralytic idiocy. The determinate causes or pathological conditions rendering the child idiotic are lack of development or of nutrition, or disease or injury affecting the brain either before or after birth.

The following classes of idiocy are recognized by Ireland:

*Genetous idiocy*, cases which cannot be traced back to any known specific disease, and whose pathology cannot be properly diagnosed until after death. The condition of mental deficiency is *complete before birth*, and a neuropathic family

history or some one of the conditions above mentioned is usually ascertainable to account for the direct hereditary transmission of the disease.

An interesting developmental abnormality common in the subjects of genetous idiocy is the high-vaulted and narrow palate. The mental state of the idiot may be said to remain at the status of the infant, or very slowly move toward the maturer state of the adult, never, of course, attaining a high degree of perfection.

The expression of the idiot is generally good-natured and confiding; the head is not necessarily small, although irregularity of formation, flatness in the occipital region and a rapid slope of the clivus are often present. The early symptoms of genetous idiocy are constant sleeping in early infancy and absence of interest and attention to its surroundings, inability of the infant to suckle well, a feeble grasp, failure to react to sensory impressions and sight, and backwardness in walking and talking. The occurrence of such symptoms in the presence of a vitiated heredity should always arouse our suspicions. Genetous idiocy forms the largest class of all cases of idiocy, and the prognosis is better than in those cases in which the child has been born with full possession of his brain-power, and has afterwards been deprived thereof.—(LANGDON DOWN.) Varieties of genetous idiocy are the *Mongolian type*, so-called from the resemblance these children bear to the Mongolian race, and the *amaurotic type*, first described by Sachs. In the latter variety the infant is apparently healthy at birth, but in the course of a few months it begins to droop and manifest signs of mental and physical breakdown, with amaurosis. The outcome is fatal.

*Microcephalic* and *hydrocephalic idiocy* are forms of idiocy which are usually congenital, like genetous idiocy, although hydrocephalus, with its consequent baneful effect upon the intellect, may not develop until a later period.

*Eclampsic*, *epileptic* and *paralytic idiocy* belong to the acquired forms of the disease, developing in association with



Other disorders of the nervous system. The first variety includes those cases in which convulsions were prevalent during the dentition period, from which the child has recovered but the brain structure has not escaped permanent nutritive impairment. These cases are frequently mutes, or they are afflicted with speech impediments.

*Epileptic idiocy*, as the name implies, is mental deficiency, resulting directly from epilepsy. As epilepsy is one of the commonest causes of idiocy and insanity, epileptics will either manifest idiocy if the mental faculties are early impaired, or epileptic dementia if the baneful influence be delayed beyond the period of childhood.

*Paralytic idiocy* depends upon destruction of cerebral substance from lesions which may have developed either before birth (congenital idiocy) or after birth (acquired idiocy). In these cases there is frequently sufficient asymmetry of the brain present to produce noticeable inequality of the skull. Hemiplegia, more or less complete, the arm usually more affected than the leg, diplegia, or simply paresis of certain muscles, and imbecility, are the accompanying conditions.

*Inflammatory idiocy* includes those cases following meningitis or some of the infectious fevers (post-febrile insanity) and idiocy depending upon atrophy and hypertrophy of the brain, the result of inflammatory changes.

*Sclerotic idiocy* presents sclerosis with atrophy of the brain, diffuse sclerotic changes, and glioma with sclerosis (WILMARTH, *Alienist and Neurologist*, Oct., 1890). As predisposing causes are mentioned the tuberculous diathesis; neuropathic heredity; alcoholism. Accidents to the mother during pregnancy and traumatism to the child's head during or after birth are exciting causes.

*Syphilitic idiocy* is not considered a very common form, and, when present, usually takes a downward course, placing it more closely with dementia than with idiocy. Without producing idiocy, however, syphilis not infrequently renders children backward both mentally and physically, as the phenomenon of *infantilism* so clearly demonstrates.



*Traumatic idiocy* results from pathological changes in the brain, induced by a destructive injury. A certain amount of inflammatory action must always be taken into consideration in these cases, but the effects of the injury predominate over those of the inflammation. Naturally, a certain degree of relationship exists between the character of an injury or the location affected and the degree of idiocy to be anticipated therefrom.

*Idiocy by deprivation* is that condition of mental inefficiency resulting from the absence of two or more of the special senses. In such a case the brain may be perfectly normal and the faculties unimpaired, but the unfortunate deprivation of both the sense of hearing and of sight through such diseases as ophthalmia and scarlet fever occurring at an early period of childhood will result in complete mental obtuseness unless proper educational training be instituted. In some instances children have been born deaf and blind.

*Deaf-mutism* stands as an independent condition, the result either of an acquired deafness through scarlet fever, typhoid fever, meningitis (especially epidemic cerebrospinal meningitis) and otitis media, or as a congenital deafness, on account of which the child does not learn to talk. If acquired after the seventh year, the child usually escapes mutism. Acquired deaf-mutism has, therefore, no relation to idiocy, but congenital deafness is a common symptom of idiocy, simply indicating one phase of the hereditary nervous deficiency of hereditary idiocy. Then, again, idiotic tendencies in a child are markedly increased by the absence of or obtuseness of any of the special senses, so that acquired deafness is a most serious calamity to befall a child of this stamp.

**Cretinism; Cretinoid Idiocy, or Sporadic Cretinism.**—Cretinism is an endemic condition prevalent in certain mountainous regions of Europe, especially inclosed in valleys. The abnormal mental and bodily development is associated with goitre, and cretinism is only found where goitre is prevalent. The impaired function of the thyroid is accepted as the cause for these manifestations.

The *symptoms* of cretinism are short stature; mental deficiency: loose, flabby skin; depression of the root of the nose and great distance between the eyes; obtuseness of hearing or deafness, and goitre.

*Sporadic cretinism*, or *cretinoid idiocy*, is a condition of myxœdema resulting from absence of the thyroid gland. This condition was first described by Fagge (*Medico-Chirurgical Transactions*, London, 1871), and numerous cases have since been published both in America and Europe.

The child may be born without a thyroid from embryonic degeneration of the same, or its degeneration may not begin until after birth. This has apparently resulted, in some instances, from an acute illness.

The first symptoms to be noted are apathetic dulness and a large, thick tongue. These signs may show themselves in early infancy or not until the child is several years old. The growth becomes stunted, the hands and feet are short and bumpy, the skin loose and wrinkled; temperature subnormal, and the ossification of the cranial bones is delayed; the head is large, fontanelles open, the nose flat and eyes widely separated, the enlarged tongue protrudes slightly from the mouth and the lips are thickened. Altogether they present a characteristic picture—a dwarfed, ugly, usually sluggish creature. The remarkable feature of this form of idiocy is the prompt improvement produced by thyroid treatment.

The *treatment* of idiocy in general is one of training, which cannot be entered upon here. The child's health must be kept in the best possible state, and, as the unhampered action of the senses is most important, adenoids and enlarged tonsils must be removed when they interfere with hearing and nasal respiration. There are special schools where proper training of the intellect through the various senses is carried out. Public institutions are hampered by overcrowding. In some of the private schools the results obtained have been far beyond expectation in many cases.

*Cretinoid idiocy* is the only form in which positive results

are to be expected from medicinal treatment. The *dessicated extract of thyroid gland* is administered in doses ranging from  $\frac{1}{2}$  m a half-grain twice daily in the beginning to one to two grains  $\frac{1}{2}$  ns thrice daily later, in the absence of unfavorable symptoms  $\frac{1}{2}$  ns. Relapses usually occur on discontinuing treatment. The  $\frac{1}{2}$  ns confusion of cases of rickets with cretinism has led to the public  $\frac{1}{2}$  ns lication of cures by other therapeutic means.

For the various other forms of idiocy, constitutional treatment is often indicated, especially anti-tuberculous and anti-syphilitic treatment. Besides, such remedies as *Baryta carb.*, *Calc. phos.*, *Aurum*, *Kali phos.* and *Sulphur* undoubtedly exert a favorable influence upon the growth and mentality of backward and imbecile children, but, unfortunately, nothing very tangible can be expected.

#### DISEASES OF THE BRAIN AND ITS MEMBRANES:

##### ACUTE LEPTOMENINGITIS.

Acute inflammation of the pia mater is in most instances secondary to some one of the infectious diseases, notably pneumonia, typhoid fever, scarlet fever and influenza. An unquestionable relationship seems to exist between many cases of meningitis and entero-colitis, the so-called metastasis of the latter condition to the brain, observed by our older clinicians, being explained by the possibility of infection of the meninges with pathogenic bacteria from the alimentary tract, or by the direct action upon the brain of the toxins generated there. Beside these causes, traumatism, sunstroke and acute nephritis also may induce a meningitis, and suppurating otitis media, erysipelas of the scalp and abscess of the brain are dangerous in that they invite extension of the infection to the meninges.

The epidemic variety of meningitis, usually described as *cerebro-spinal meningitis* or *spotted fever*, from involvement of the meninges of the cord and the appearance of an exanthem during its course, is of infectious origin, the *diplococcus intracellularis meningitidis* of Weichselbaum being the specific

micro-organism (see "Acute Infectious Diseases"). Purulent meningitis from infection with the *pneumococcus* may also develop at times apparently primarily.

The *pathological changes* observed in the brain of children dying of meningitis vary with the severity of the case and stage at which a fatal termination took place. Frequently nothing more is found than intense hyperæmia and œdematous infiltration of the pia mater. Such cases run a rapid course, and the diagnosis is frequently not made *intra vitam*. When, however, the onset has been more gradual and the duration longer, large flakes of fibrin and islands of purulent exudation are found covering the convexity of the brain and filling-in the convolutions, notably over the anterior lobes. In many instances the membranes of the cord are simultaneously involved, notably over its posterior surface. This involvement is especially marked in the epidemic variety and in purulent meningitis with spinal symptoms.

The *symptomatology* of acute leptomeningitis points to involvement of the convexity of the brain as a predominating condition. It is most common in infants, in whom it may develop idiopathically, or in conjunction with an enterocolitis or broncho-pneumonia. I recently saw such a case, which was undoubtedly toxic in origin, the autopsy revealing a large caseous mass in the stomach consisting of a conglomeration of bread and milk. During the course of an apparently simple gastro-enteritis this infant was suddenly seized with convulsions and rapidly-rising temperature, death resulting within twenty-four hours, from meningitis.

There is always danger of meningitis in cases of purulent *otitis media* in which, owing to the proximity of the dura mater to the seat of the pus, the thinness of the temporal bone and the channels of communication existing in childhood between the dura and the middle ear. For this reason acute purulent meningitis, cerebral abscess and thrombosis of the jugular bulb must always be borne in mind as possible complications.

*Infantile acute leptomeningitis* may not be suspected until

convulsions set in, but in many instances the child will present definite symptoms pointing to the beginning of a meningeal inflammation, *viz.*: bulging of the fontanelle; malaise; elevation of temperature; irregular or contracted pupils; strabismus; projectile vomiting and rigidity of the cervical muscles. As the case progresses, coma; dilated pupils; convulsions, and death or ultimate recovery, with impairment of the special sense, may ensue.

The meningitis occurring later in childhood is almost invariably secondary to an infection or septic condition with the exception of the *epidemic form of cerebro-spinal meningitis*, and *tuberculous meningitis*, the later variety, of course, being in many instances but the terminal event in a more or less general tuberculosis (see Tuberculous Meningitis). A secondary meningitis is naturally masked more or less by the disease which it accompanies, and if the lesions are confined solely to the convexity it is difficult of recognition, as the symptoms of stupor, delirium, convulsions, dilated pupils, irregular respiration, irregular and slow pulse and vomiting might be attributed to the original disease. Unless occurring in children over two years old, the characteristic slowing of the pulse and the Cheyne-Stokes respiration may be poorly developed, although the majority of cases I have seen even in infants presented this symptom. Meningitis as a complication of another acute illness becomes a serious matter, death supervening, within a few days of the onset in convulsions or coma, the temperature often running very high. If the process has extended to the base and into the spinal canal, there will be added opisthotonos; strabismus; deafness and cutaneous hyperæsthesia.

The *prognosis* of meningitis is always grave. It is conceivable that mild cases, such as may accompany bronchopneumonia, enterocolitis, or result from traumatism, frequently recover, but the question of a correct diagnosis arises here. The likelihood of idiocy and permanent sensory and motor defects remaining after meningitis should not be lost sight of.

In the **diagnosis** of meningitis the only positive signs are those indicating actual compression or continued irritation of the cerebral structure, manifesting themselves as contracted, and, later, dilated, pupils; ocular palsies; opisthotonos; coma; projectile vomiting; headache; high fever, with irregular and slow pulse, and irregular breathing and localized paralyses or convulsions. Although such symptoms are progressive prostration; elevated temperature; bulging of the fontanelle; squinting; projectile vomiting and convulsions are strongly suggestive of meningitis, still they may all be induced by even harmless ailments which have no connection at all with meningitis, being purely toxic and coming under the category of "teething," "reflex irritation," "worms," and the like, or marking the advent of some acute illness. Cerebral symptoms coming on suddenly and accompanied by high fever are *toxic* in origin with greater likelihood than inflammatory. They usually pass off in the course of a few days—with the crisis of a pneumonia; after emptying the bowels in an acute gastro-intestinal intoxication or with the appearance of the rash in an exanthem. On the other hand, cerebral symptoms coming on insidiously or appearing in the later stages of an attack of pneumonia, influenza, ileo-colitis, are of graver import, and as a rule prove to be due to meningitis. In *autointoxication* there is absence of high fever and diacetic acid and acetone may be found in the urine. *Uræmia* and *diabetic coma* must also be thought of.

#### TUBERCULOUS MENINGITIS; BASILAR MENINGITIS.

Tuberculous meningitis is often spoken of as *basilar meningitis* on account of the constancy with which the tuberculous lesions develop at the base of the brain, they, only in exceptional instances, involving the convexity, and then as an extension of the process from the base. The opposite condition holds good in simple meningitis; but all cases of basilar meningitis are not necessarily tuberculous, some being of syphilitic origin and others a primary infectious disease ex-



existing without the presence of any inflammatory lesions elsewhere excepting an exudative process about certain tendons and sheaths. This form of meningitis (posterior basic meningitis) is not uncommon in infants, and is frequently confounded with tuberculous meningitis. Still (*The Micro-Organism of Simple Posterior Basic Meningitis in Infants*, *Trans. Brit. Med. Ass.*, 1898) has demonstrated a *diplococcus* in these cases. Koplik (*Amer. Jour. Med. Sciences*, February, 1905) finds that the cases described by Still, while usually sporadic, also occur during epidemics of cerebro-spinal meningitis and are due to the *meningococcus intracellularis*. Koplik describes a variety occurring in older children which may be complicated with pneumonia or be secondary to the same.

Tuberculous meningitis is by no means a rare disease. It is quite common for children of the tuberculous diathesis to become thus affected, and the victims of general tuberculosis frequently die of a terminal meningitis. This is especially so during infancy, while in older children the meningitis may exist as an apparently primary condition; but even here an autopsy usually reveals unsuspected tuberculous lesions of the internal viscera. Tuberculosis of the bones and lymphatics is also a strong predisposing factor to tuberculous meningitis. Heredity plays an important rôle, and in certain families the disease appears with appalling regularity. Furthermore, all conditions favoring or resulting in malnutrition offer a predisposition to the disease.

Primary meningitis in infants is most often tuberculous. The tubercle bacilli may gain access to the meninges through the nose by means of the vein of Zuckerkandl or through a necrosed cribriform plate. I had under my observation an infant in whom tuberculous meningitis followed upon an attack of purulent coryza that was treated with injudicious local applications and probing.

The pathological changes found in the brain are mainly tubercles situated along the course of the blood-vessels at the



base of the brain, chiefly following the sylvian artery; inflammatory reaction in the pia with the production of lymph- and pus-cells, and exudation into the ventricles (acute hydrocephalus), and more or less infiltration of the brain substance (meningo-encephalitis). The blood-vessels are injected and bathed in a sero-gelatinous exudate. In the advent of much effusion the convolutions appear flattened, but the amount of effusion and exudation seen in simple meningitis is rarely present. Ordinarily the eruption of tubercles is limited to the base, for which reason tuberculous meningitis is also known as basilar meningitis, but tuberculous deposits may also occur in atypical localities producing focal symptoms—cerebral tuberculosis—or these deposits may simply accompany basilar meningitis as an encephalitis. The clinical manifestations depend upon the direct pressure of the tuberculous deposit upon the cranial nerve roots at the base of the brain as well as upon increased weight of the brain and intracranial tension from the exudation of serum. The meninges of the cord at its site of origin are almost invariably involved; this is the cause of the retraction of the head characteristic of the disease.

**Symptomatology.**—For physiological reasons the symptoms will vary with the age of the child. This also modifies the clinical course of the disease to a certain extent, as tuberculous meningitis is often but the terminal event of a more or less general tuberculosis. There is, however, a sufficiently large number of cases of tuberculous meningitis occurring without demonstrable evidence of pre-existing tuberculosis to justify us in suspecting every primary meningitis in a young child, in the absence of an epidemic of cerebro-spinal fever, as being tuberculous.

The characteristic slowing of the pulse and the disturbed rhythm of the respiratory act are generally not observed until after the second year, as the inhibitory centres are not fully developed until this time. So also, intellectual and sensory disturbances cannot be obtained until the child's brain is correspondingly developed.

A typical case pursues the following course: For a few weeks the child manifests signs of indisposition and malaise. It loses interest in its games and associates, the appetite fails, and emaciation and progressive anæmia become noticeable. There is constipation, and at times a slight elevation of temperature. As the cerebral lesions become of sufficient prominence to produce specific symptoms, headache, vomiting and slowing of the pulse develop. They constitute the main symptoms of the first period of the actual meningitis, or the stage of cerebral irritation. Together with slowing and irregularity of the pulse there is irregularity in the respiratory rhythm, later on approaching the Cheyne-Stokes type of respiration.

Constipation is present from the beginning, the abdomen being flaccid, and in some instances noticeably retracted.

Vomiting is usually of the projectile type, after which the child seems weak and apathetic, gradually going into a state of stupor in the later stages. The vomiting results from irritation of the sensory meningeal branch of the vagus.

Vasomotor disturbances present during this period are alternate flushing and paleness of the face, and the *tache cérébrale*, a broad, red line produced by drawing the fingernail across the skin of the abdomen, persisting for a few minutes and indicating vasomotor paresis. Irregular innervation of various muscles supplied by the cranial nerves is also a common symptom of this stage. To these manifestations strabismus and twitching of the facial muscles belong most prominently. There is ptosis and the eyes are often fixed in a characteristic vacant stare. The accumulation of fluid in the ventricles is largely responsible for the pressure symptoms upon the centres of the oculo-motor nerve. Likewise, the increased weight of the brain causes it to sag down upon the base of the skull and, by direct pressure upon the abducens, set up an inward deviation of the eyes. The deposit of miliary tubercles and inflammatory exudate upon the basal nerve roots tends to produce paralysis in the parts supplied by them.

As the vagus and glosso-pharyngeus become involved death is the inevitable result.

Retraction of the head, opisthotonos, rigidity, twitching and automatic movements of extremities belong to the spasmodic manifestations of tuberculous meningitis. The most characteristic of these is the retraction of the head, which is a strong presumptive sign of basilar meningitis, although it is frequently seen in toxæmia. We must also bear in mind that rigidity of the neck need not be continuously present during the course of the illness, and I have seen cases where it was either altogether absent or only slightly developed.

In infants emaciation is pronounced and progressive. Convulsions may occur, but they are not a constant feature. Likewise, the shrill, piercing cry, "*cri hydrocephalique*," may or may not be heard.

The case now gradually goes over into the second period, that of paralysis. The duration of the first period is variable, the average being from ten days to two weeks. Its course is also irregular, unexpected signs of improvement frequently showing themselves, from which the child soon lapses back into its original state.

The second period is characterized by progressively increasing stupor, increased frequency of the pulse and failing heart, dilatation of the pupils, opisthotonos with general relaxation of the muscles of the extremities in which transient convulsive movements may be noted, complete coma and death. Although the temperature is not high during the course of the disease, unless an associated tuberculous process renders such the case, still there may be a rapid rise shortly before death, which may attain to a high degree of hyperpyrexia. Death in convulsions is not common. The average course is from two to three weeks, not including prodromal symptoms. A case of meningitis living beyond six weeks may safely be looked upon as non-tuberculous.

The *prognosis* is always unfavorable. I have seen a few apparently genuine cases recover, but I doubt if they will be

spared a relapse or death from general tuberculosis at some later period, if they really were tuberculous. Unfortunately, an absolutely positive diagnosis cannot always be made, which leaves room for doubt in those cases going on to recovery. The toxic cerebral symptoms developing during pneumonia in childhood so closely resemble tuberculous meningitis in many of its phases that it really becomes difficult at times to estimate their true significance. They are certainly within the reach of our remedies, as I have repeatedly satisfied myself, but the remedies which control them fall short in the true tuberculous process.

The *diagnosis* of tuberculous meningitis is based upon its gradual onset, the presence of the tuberculous diathesis or tuberculous family history, and the development during the first period of the characteristic symptoms, namely, constipation, headache, slowing of the pulse, vomiting and drowsiness. Later the condition becomes unmistakable, but in the early stages we must exclude the acute infectious diseases or acute gastritis with cerebral symptoms.

*Simple meningitis* is differentiated by its rapid onset and acute course; *cerebral hyperæmia* by its transitory nature and *hydrocephaloid* by its association with diarrhœal or other exhausting diseases. The last-named condition is described in another chapter. (*Diseases of the Intestines*, p. 206.)

The positive recognition of meningitis is made possible by means of *lumbar puncture*. This procedure not only verifies the diagnosis of meningitis, but enables us at the same time to recognize its true nature, *i. e.*, whether tuberculous or purulent.

#### LUMBAR PUNCTURE.

Lumbar puncture was introduced by Quincke as a method of diagnosis in intracranial affections and its value in this direction is now firmly established. Besides it has also attained to some extent the role of a therapeutic agent. Owing to the continuity of the sub-dural space throughout the entire

cerebro-spinal nervous system, it is self-evident that a specimen of fluid withdrawn from the lower end of the dural sac is identical in character with the fluid high up in the spinal canal, and even within the cranium. Clinical experience has proven this to be true so regularly that now we are in a position to learn the nature of an intracranial effusion or accumulation with practical certainty. Furthermore, by means of a canula inserted into the spinal canal we can estimate the intracranial pressure as readily and accurately as, for example, we estimate the blood pressure in the peripheral arteries.

Before entering into a discussion of the character of the cerebro-spinal fluid under normal and abnormal conditions, it will be well first to describe the technique of performing lumbar puncture.

We must remember that the spinal cord proper terminates in the conus at the second lumbar vertebra, where it divides into two coarse strands of fibres, which hug the lateral walls of the spinal canal. These bundles constitute the cauda equina and there is plenty of space between them for the safe introduction of a canula; besides, they are more or less movable and therefore not readily wounded.

If, therefore, we introduce a small trocar between the spines of the third and fourth or fourth and fifth lumbar vertebrae, we enter the dural sac most satisfactory for the purpose of aspiration.

The best instrument to use is the original Quincke needle, which is made by Tiemann & Co., after a pattern brought to this country by Koplik. An aspirating needle—10 cm. long and 1 mm. in diameter—answers in the case of children, but a small trocar will be found more convenient to handle. The operation must be performed under the strictest asepsis; this applies to the operator's hands, the instrument, and to the skin at the site of puncture. Scrubbing with soap and water, followed by the use of alcohol and lastly a 1-1000 solution of bichloride is to be recommended.

It is by no means easy to locate the different vertebrae by

attempting to count them from above downward, but if we remember that a line drawn across the back on a level with the crests of the ilia will intersect the fourth lumbar interspace, it is a simple matter to select either this space or the one above it as the site for puncture. We may puncture as high as the second interspace, but there is not only an imaginary, but an actual advantage in selecting the lowest point, for as Sahli was able to demonstrate, pus and other elements tend to gravitate to the lowest point, and when present in inconsiderable amount, clear fluid may be withdrawn from the second, while a cloudy one may come from the fourth interspace.

The patient is laid upon the right side, and the spinal column bowed as much as possible by flexing the legs upon the abdomen and pressing down upon the buttox, at the same time bending the upper portion of the back by downward pressure upon the shoulders. Care should be exercised not to exert pressure upon the neck, but always upon the shoulders. The spines of the vertebræ now stand out prominently and we are in a position to plunge between them into the canal. When the patient is comatose no anæsthetic is required, and when partly conscious ethyl chloride should be used locally. In young children the laminæ of the vertebræ are horizontally placed and the interspinous ligament is not very firm. For this reason we can pierce directly between the spines and enter at a right angle to the spinal column. In older children the laminæ are somewhat overlapping and the interspinous ligament is tough and firm. Here it is best to pursue the course first recommended by Quincke, namely, place the point of the needle to the lower side of the median line and a little below the interspace; then pierce upward and inward, thus avoiding the ligament and at the same time slipping in between the laminæ. In a child two years old the dural sac is penetrated when the needle is inserted for a distance of from 2 to 3 cm., in adults it must penetrate 4 to 6 cm. With a little practice we soon learn to recognize when the needle is in the spinal canal: there is no further resistance and the point can be freely

moved. The stilet of the trocar is now removed and the first few drops of fluid are allowed to flow out; the remainder is caught in a sterilized graduate, in order to estimate the quantity withdrawn. Ten cc. is sufficient for diagnostic purposes, but when the pressure is great we may withdraw as much as fifty cc. A portion of this can be used for making cultures

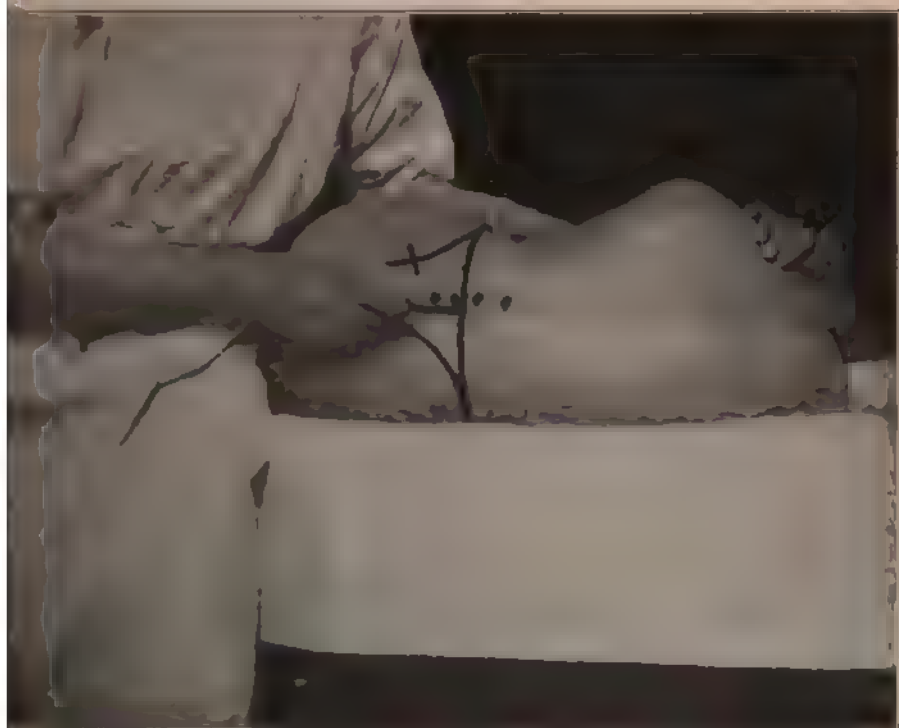


FIG. 47. METHOD OF PERFORMING LUMBAR PUNCTURE.

or for inoculating guinea pigs. The balance is studied macroscopically and microscopically. The chemical examination is also important.

The study of intracranial pressure is interesting, but of little clinical value in pediatric practice. For practical purposes we can estimate this sufficiently by the force with which



the fluid flows from the canula. If a manometer be attached to the canula the pressure can be measured in mm. of mercury. The normal pressure in adults in the prone position is 5 to 7.3 mm. Hg.; a pressure above 15 mm. Hg. is indicative of conditions such as meningitis and brain tumor (SAHL). Koplik gives the pressure as from 5 to 35 mm. of mercury. It is lower in children than in adults.

Under normal states of pressure the fluid comes from the canula drop by drop. When the pressure is increased the drops come more rapidly and with considerable effusion it will spurt out in a stream. The stream is not steady and is affected by respiration. It is interesting to note the immediate amelioration of symptoms when such pressure is relieved, but unfortunately this improvement is but temporary in the majority of instances. In young infants the fontanelle offers an additional means of estimating intracranial tension.

The normal cerebro-spinal fluid is clear, colorless and limpid. Its specific gravity is very little above that of water, from 1003 to 1005. Besides a low percentage of albumen (less than  $\frac{1}{4}$  per cent.; LENHARTZ), it contains a trace of sugar and salts. Alfred Hand (*Phila. Med. Journal*, August, 1902), found from 0.5 to 1.9 per cent. albumen by bulk with the potassium ferrocyanid test, while in ordinary meningitis there will be from 4 to 5 per cent. and in tuberculous meningitis it ran as high as 16 per cent. in one of his cases.

Absence of sugar speaks for ordinary meningitis, while its presence favors a tuberculous condition (HAND).

The admixture of blood may be due to the wounding of vein and rather spoils the specimen for gross and microscopic study. It may, however, throw light upon the diagnosis when hæmorrhage into the cord or ventricles is suspected.

In tuberculous meningitis the fluid is usually but a trifle cloudy; in fact, it may require close inspection in a good light before we will recognize a slight turbidity. Such fluid should be placed in a conical sediment glass in a refrigerator for 24 hours, at the end of which time a delicate mesh-work of spon-

taneously coagulated fibrin will have formed. Toward the latter part of the disease the fluid becomes more cloudy.

In epidemic cerebrospinal meningitis the fluid may be clear in the early stage, but later it becomes purulent. The same may be said of the other forms of meningitis, usually secondary—in the early stages of which there may be clear fluid with suspended flakes of fibrin, just as we see within the cranium—later becoming creamy or admixed with blood.

In hydrocephalus the fluid is clear, although it contains some leucocytes.

The cellular elements of the cerebro-spinal fluid are of definite significance. As in any other pyogenic infection the exudate consists chiefly of polynuclear leucocytes in suppurative meningitis. This also holds good in meningitis due to the *pneumococcus*, and the epidemic variety due to the *meningococcus intracellularis* of Weichselbaum.

French writers lay great stress upon the predominance of lymphocytes in tuberculous meningitis. While the value of cytodagnosis in cerebral inflammations is not without limitations, still a marked increase in lymphocytes over polynuclear elements is strong presumptive evidence in favor of a tuberculous infection. Osler states that in his recent cases he was not able to verify these claims and it is quite true that the lymphocyte count is subject to a wide range of variations. Thus, Hand found in a case of tuberculous meningitis that one tapping gave 35 per cent., while a subsequent one gave 85 per cent. lymphocytes. A persistent excess of polynuclears seems to rule out a tuberculous infection with more or less certainty. Again, the number of tubercle bacilli present seem to bear a direct ratio to the number of lymphocytes that will be found in the specimen.

In purulent exudates ordinary cover glass preparations stained with methylene blue are sufficient for the study of the bacteria present. Streptococci and pneumococci are recognized by their morphology while the micrococcus of epidemic cerebro-spinal fever is a diplococcus similar in ap-

pearance to the gonococcus. The majority of these diplococci will be found within the pus cells. As Park puts it, the cells are crowded with the diplococci. The differentiation between meningitis due to this diplococcus and that due to pneumococci or streptococci is of great clinical value, as forty per cent. of the former cases get well, while almost all of the others die.

Tubercle bacilli are as a rule present in scant numbers, but if we allow the fluid to stand for twenty-four hours, the bacilli and cellular elements will become entangled in the meshwork of fibrin, which has formed at the end of that time. The fluid can then be centrifuged and the sediment stained for tubercle bacilli in the usual manner. By this method their demonstration becomes successful in the majority of cases.

The indications for the lumbar puncture are any obscure cerebral condition in which there is clinical evidence of inflammation of the meninges or the presence of an exudate, whether it may be serous, purulent or hæmorrhagic.

So far its chief value has been that of a diagnostic aid, the importance of which cannot be questioned. Temporary relief of symptom, coma, convulsions, has also attended its use, and in the control of uræmic convulsions and coma it has proven of value. Complications, such as pneumonia, contraindicate it. When carried out *lege artis*, and for a definite purpose, lumbar puncture is not only justifiable, but absolutely essential to the scientific practitioner.

**Treatment.**—The general management of cases of acute leptomeningitis and tuberculous meningitis differs somewhat, owing to their clinical diversity. Leptomeningitis runs an acute course and frequently exists as a complication of another acute illness, for which reason it becomes more difficult to manage than the subacute tuberculous type. Hyperpyrexia may be present, necessitating frequent sponging, and the occurrence of convulsions calls for a warm bath in infants or a warm pack in older children. Should these mani-

fest a tendency to recur or become persistent, a hot mustard pack is more efficient. Stimulation may become necessary in the later stages. The ice-cap is recommended by some, but I doubt its efficacy.

In tuberculous meningitis, prophylaxis offers the only promising results. Aside from a low diet, attempts at keeping the bowels open by means of enemata and the employment of simple dietetic measures, beside stimulation when indicated, there is little to be done with the exception of what medicines may accomplish.

*Acon.* may be indicated early in primary cases of leptomeningitis.

*Apis.*—This is one of the most important remedies in meningitis, both the subjective symptoms, as well as the pathological tendency of the drug, indicating it in many instances. Particularly characteristic of *Apis* is the shrill, piercing cry, which is usually heard at night, while the child is asleep or in a soporose condition. Other prominent symptoms are difficulty in swallowing; retraction and rolling of head; strabismus and dilated pupils; gritting of the teeth; suppressed urine; convulsions and coma. The amount of effusion is usually considerable in cases calling for *Apis*.

*Arnica* is recommended in traumatic cases.

*Belladonna* presents a true picture of the early stages of a meningitis, at which time cerebral hyperæmia is the most prominent condition. It is of little use after effusion and exudation have taken place. The face is flushed; eyes bright with dilated pupils; there is pronounced hyperæsthesia of the senses with frequent starting, especially on attempting to go to sleep; the pulse is full and bounding, the fontanelle prominent, and general convulsions set in.

*Bryonia* corresponds to the exudation stage. Fever with great thirst; and dryness of the skin and mucous membranes; irritability of temper with marked indifference and desire to remain quiet; prostration; constipation; bursting headache; face dark red; head retracted; constant chewing motion of

*Cicuta*.—For the irritative stage of meningitis *Cicuta* is a most valuable remedy, particularly when there are general convulsions, beginning with twitching in the fingers and ending in complete unconsciousness. There is rolling of the head; fixation of the eyes; boring of the occiput into the pillow. The child is greatly agitated, grasping at its mother in a frightened manner when being taken up.

*Cuprum*.—Especially useful during the exanthemata, after recession of the eruption. (*Zincum*, "not able to develop exanthemata.") The *Acetate of Copper* was first recommended by Dr. George Schmid, of Vienna, for the cerebral symptoms resulting from the retrocession of any of the acute exanthemata, or from difficult dentition, in cases not active enough for *Belladonna* (HUGHES, *Pharmacodynamics*).

*Gelsemium* may be indicated upon its well-known symptoms of drowsiness, paralysis of the muscles of the eye, convulsive movements during sleep, intense headache and low form of fever.

*Glonoin*.—Intense cerebral congestion, throbbing carotids, high arterial tension; high fever, with paleness of face or intense redness. *Veratrum viride* is indicated in similar cases, where the symptoms are intense. *Glonoin* suits most particularly the form of meningitis developing after sunstroke.

*Helleborus*.—Exudative stage. Great irritability, eyes rolling from side to side, wrinkling of the folds of the forehead as if frowning, chewing motion of the mouth, boring the head into pillow, cervical opisthotonos, automatic movements of one or more of the extremities with jerking and twitching of groups of muscles; scanty, dark-colored urine with sediment like coffee-grounds.

*Kali hydrojodicum* has been used with apparently some success to absorb the effusion. It is more particularly adapted to the epidemic variety of meningitis, in which cases, as matter of fact, more can be done than in the other forms of meningitis. It is generally used in appreciable doses.

*Mercurius*.—Intense headache, as if compressed by tight

band. The child is drowsy, cries out in its sleep and tosses about restlessly. There may be a clammy offensive sweat, foul odor from mouth, thickly-coated and swollen tongue. *Mercury* is one of the most useful remedies to remove the inflammatory exudate.

*Opium*.—Sopor; pupils contracted or immovably fixed, with glassy, half-closed eyes and pale face; stertorous breathing.

*Stramonium*.—Violent delirium; face red and bloated, with wild expression of eyes; automatic movements of hands and feet, convulsions and coma. Child awakens from sleep screaming and terrified.

*Sulphur*.—On account of its pronounced absorptive action *Sulphur* is frequently indicated in the later stages of meningitis, especially when the case comes to a standstill. Many of its characteristic symptoms are frequently present in these cases, but even without such the above condition fully justifies its use.

*Zincum*.—Continuous movement of the lower extremities, particularly the feet; profound nervous depression, abolition of reflex action; meningitis developing with the exanthemata or during an epidemic of such, when the rash recedes or does not make its appearance.

In tuberculous meningitis *Iodoform*, *Lycopodium*, *Calc. carb.*, *Calc. phos.*, *Spongia*, *Apis* and *Sulphur* seem most applicable. The alleged efficacy of *Iodoform* has attracted considerable attention of late. Several cases have been reported cured, both by the internal administration of the drug in the 2x to 6x trit. and by the use of an *Iodoform* salve as an inunction, after shaving the scalp (BAILEY, *Medical Counselor*, June, 1898). I have in several instances relieved meningeal symptoms accompanied by retraction of the head, occurring during the course of acute pulmonary affections, and in one case in a syphilitic infant, with this drug, but they were most probably of toxic origin. The provings of *Iodoform* contain many symptoms strongly suggesting its use in meningitis (see *Cyclopædia of Drug Pathogenesis*, vol. iii).

*Lycopodium* is well adapted to many cases coming on insidiously in strumous children. Goodno (*Practice of Medicine*) reports such a case cured by *Lycopodium* 6x.

*Spongia* is of great importance on account of its relationship to scrofulosis and tuberculosis (HERING). Its chemical composition would suggest a similarity with *Iodoform*.

#### HYDROCEPHALUS.

Hydrocephalus is a chronic idiopathic disease, in which there is an excess of serum in the cranial cavity (hydrocephalus cerebri). The so-called *acute hydrocephalus* is in reality tuberculous meningitis.

There are two forms of chronic hydrocephalus, namely, *external hydrocephalus* and *internal hydrocephalus*. The former consists in the accumulation of serum between the dura mater and the arachnoid. It is rare, almost invariably occurring secondarily to a congenital defect of the brain, meningeal hæmorrhage, pachymeningitis, or atrophy of the brain. In the last instance the serous effusion occupies the space left vacant by the deficient brain, and it is spoken of as *hydrocephalus ex vacuo*. External hydrocephalus is also encountered congenitally. When not associated with a hopeless intracranial condition it presents the best prospects for cure by operative measures.

**Chronic Internal Hydrocephalus** is the commonest form of the disease, and in speaking of hydrocephalus this is the variety usually implied. The largest number of cases are congenital. The head may be so large at full term as to impede delivery, or the effusion be but trifling and accumulate so slowly that the head does not become noticeably enlarged until several weeks after birth, making it difficult to determine whether the case was congenital or acquired (MILES). Again, the child may be perfectly healthy at birth and during the early months of infancy, no enlargement of the head occurring until the fourth month, or even later.

Internal hydrocephalus is almost invariably a primary cor



tion. In rare instances it is found associated with tumors or inflammatory processes at the base of the brain when such conditions cause obstruction of the foramen of Magendie or obliterate the communications between the ventricles of the brain. Accumulation of cerebro-spinal fluid in the ventricles may result from diminished resistance of the cranial walls, and also from causes directly increasing the blood-pressure in the brain, *i. e.*, whooping-cough, bronchitis, emphysema and convulsions; indeed, a history of convulsions can be obtained in most cases. Syphilis and rickets, by inducing malnutrition of the osseous system and anæmia, may act as predisposing causes. Some cases are distinctly inflammatory, the process attacking the ependyma of the ventricles and of the choroid plexus. In these cases the fluid is turbid, containing blood and pus cells. The cause of the inflammation is difficult to determine. A syphilitic history can be obtained in some instances, and in others a hereditary predisposition seems to exist.

The amount of fluid is sometimes enormous; it may amount to several pints.

The brain is greatly distended, the convolutions become obliterated, and the cortex may become a mere shell or the brain appear as a large cyst. The cranial bones are thin and the sutures widely separated. In rare instances premature ossification of the cranium occurs with hydrocephalus. Spina bifida and other congenital defects may be found associated.

The *symptoms* of a typical case are mainly objective. The head is rounded, its size much out of proportion to the rest of the body and in its relation to the development of the face, and the fontanelles and sutures are wide open and tense. In external hydrocephalus the enlargement is usually not so pronounced, and when the skull is still soft fluctuation can be elicited over the head.

It is necessary to remember the normal circumference of the head at different periods of infancy in order to determine whether the head be abnormally large. At birth the circum-

ference should be about fourteen inches, and at the end of the first year nineteen inches. Beside this, the relationship of the circumference of the head to the chest is important to bear in mind, the circumference of the head at birth exceeding that of the chest by half an inch; later, during the entire period of infancy, the two measurements are practically equal. From these data it is easy to determine an abnormally developed head. Every cranial enlargement does not, however, indicate hydrocephalus, the most important condition to be differentiated being rickets.

Hydrocephalus may occur *without enlargement* of the head. In such cases there is either premature ossification of the skull or a late onset of the disease. They are generally idiots and die early (HOLT). It is impossible to recognize this condition during life.

The rate at which the head enlarges varies greatly; the earlier and more rapidly the enlargement develops the more serious the prognosis. Cerebral symptoms are slight, often entirely wanting. The development of the child is, however, much retarded, and the majority of cases die early of marasmus. Those surviving this period die in early childhood, as a rule, from some intercurrent disease. The mind becomes affected and many are idiots. They are irritable and often show evidence of violent temper. In others, again, the intelligence is but slightly interfered with and they live beyond the proscribed period, being, however, entirely helpless owing to their enormously sized heads.

The *differential diagnosis* between hydrocephalus and rickets should present no difficulties, excepting when the two conditions co-exist. This is quite rare, but even then the hydrocephalus soon takes the upper hand, the rachitic condition falling in the background.

As has been said above, the hydrocephalic head is enlarged out of all proportion to the rest of the body, and presents a regular rounded outline. The root of the nose is prominent and the eyes are deflected downward, so that the lower lid

es the iris higher than normal. The face is small in comparison with the head. The fontanelles are bulging and tense, and the sutures widely separated, while the cranial bones feel thin. Fig. 48 represents a case of moderate severity in which the differentiation from the rachitic head is early shown. In rickets, on the other hand, the head is large and the centres of ossification in the frontal and parietal bones are hypertrophied. The skull is hard excepting in the occipital region, where craniotabes may be present.



Fig. 48. — A CASE OF HYDROCEPHALUS DEVELOPING SEVERAL MONTHS AFTER BIRTH, SHOWING THE EARLIER PERIOD OF THE DISEASE.

enlarged epiphyses and deformities in the extremities are associated.

The treatment is unsatisfactory. So far, surgical interference has availed but little, and seems only applicable to the chronic form. Spontaneous cures by discharge of the fluid through the nose and scalp are on record.

Tapping the head may be tried as an adjuvant, but it should be done with caution. Bartlett (*GOODNO'S Practice*) mentions the cures effected by the application of solar heat,

which is a measure that should at least be tried. "The method consists in exposing the child's occiput to the direct rays of the sun for twenty minutes each day, gradually increasing the duration of the seance until the limit of thirty or forty minutes is reached. It is believed that the local sweating acts to remove a portion of the effusion, while the thermic heat aids nutrition."

When a distinct syphilitic history can be obtained the case should be treated as one of congenital syphilis.

Grauvogl has recommended that in cases where a family tendency to hydrocephalus exists the mother should receive *Sulphur* and *Calcareo carb.* during every pregnancy, administered at suitable intervals.

The following remedies are to be studied:

*Baryta carb.*—Deficient development, both physically and mentally.

*Calcareo carb.*—"Fat babies with large heads; wide open fontanelles, which are often covered with dirty, scurfy skin; fair complexion; precocious; head sweats profusely, especially on occiput; abdomen large and sensitive to pressure, bowels inclined to be loose, feet damp and cold, dentition difficult" (C. G. R.).

*Calcareo phos.*—"Flabby, shrunken, emaciated children; skull thin and soft, with fontanelles wide open; cannot stand and do not learn to walk, want to nurse all the time; retarded dentition; emaciation with loose, green stools" (C. G. R.).

*Lycopodium.*—"Children sleep apparently soundly, but scream out suddenly in sleep, stare about and cannot be pacified" (C. G. R.).

*Kali hydriodicum* should be tried when there is a syphilitic history.

*Silicea.*—Profuse sweating about head and neck; pale, delicate children with tuberculous tendency; rickets, malnutrition of osseous system.

*Sulphur.*—Children with unhealthy skin, cutaneous eruptions, symptoms appearing after suppression of eruption, voracious appetite; old, withered look.

Other remedies that have been recommended are *Thuja*, *Psorinum* and *Tuberculinum*, and for the removal of the effusion, *Apis*, *Apocynum* and *Helleborus* are spoken of, but, as Bartlett suggests, their administration will be attended with disappointment. More reliance is to be put in the constitutional treatment.

#### CONVULSIVE AFFECTIONS.

*Eclampsia, or Infantile Convulsions.*—General convulsions occur in infancy from a variety of causes, and are among the more frequent of the nervous disorders incident to this period of life. In many respects these seizures bear great resemblance to idiopathic epilepsy, but the latter condition is not seen in early childhood, nor does it run the same clinical course nor depend upon the same etiological factors as infantile convulsions. Eclampsia may be looked upon as an explosive discharge of nerve force in the motor areas, brought about by a condition capable of suspending momentarily the normal controlling influence of the higher inhibitory centres. Such a factor may be found in *reflex irritation*, *i. e.*, sensory impulses originating in various parts of the body by being carried to the cerebrum along the afferent nerve fibres, transiently disturbing the controlling element in these centres. The modern view, however, rather favors the belief that the majority of cases are toxic in origin.

Although infancy of itself predisposes to convulsions by reason of the rapid growth of the brain and the instability of the nerve centres, still a healthy and properly cared for child rarely develops them. There is usually some constitutional disturbance, or a general disease affecting directly the nutrition of the nervous system. For this reason rickets plays such a prominent rôle as a predisposing cause.

As reflex causes may be mentioned phimosi; dentition; the presence of undigested food particles, foreign bodies or worms in the intestinal tract and retention of urine. It is doubtful whether teething, *per se*, ever produces convulsions,

and in considering the other causes it is often difficult to decide whether they act reflexly or whether they do not actually set up an auto-intoxication.

To the *toxic* cases belong those convulsions ushering in acute infectious disease, or occur as a result of indigestion; uræmia; asphyxia; jaundice; diabetes; anæmia; drugs. Lastly are to be mentioned the convulsions resulting from *direct cortical irritation* and occurring in meningitis; cerebral hæmorrhage, abscess or thrombosis; hydrocephalus; brain tumor, injury at birth.

The anatomical lesions found in children dying in convulsions are by no means constant or characteristic. The changes occurring in the brain are probably anæmic, followed by venous hyperæmia. When intense congestion, serous effusion and punctate hæmorrhages are found after death they are to be looked upon as a result of the convulsion and not as a cause of the same, death having resulted from asphyxia. The initial stage of a meningitis may also be found; or, if the convulsion depends upon organic brain disease, such a condition becomes evident.

**Symptomatology.**—Infantile convulsions are most frequently general, although a localized or partial convulsion may result as well from reflex irritation as from organic disease. In such cases the subsequent course of the disease alone will clear up the mystery, if unmistakable evidence of organic disease or reflex irritation cannot be elicited at the first examination of the case. Even a more extensive convulsion shows a tendency to begin in one extremity a few seconds before it passes to the remaining half of the body or becomes general; but the true local convulsion, or *Jacksonian epilepsy*, repeatedly commences in one extremity, and if it does not remain local, at least continues so for an appreciable time (HERTER). This form of convulsion is indicative of organic disease, the nervous discharge commencing at the seat of irritation. With it there is no loss of consciousness. Prodromal symptoms are therefore usually present, indicating the con-

mencement of a general convulsion. They may be so slight as to be entirely overlooked, or they may manifest themselves as extreme restlessness, twitching of the mouth, eyelids, extremities, and rolling of the eyes.

The convulsion proper is very similar to an epileptic fit. The child becomes suddenly rigid, the neck being thrown back, the hands clenched, with thumbs buried in the palms, and the extremities stiffen out. This stage is only of short duration, not as long as in a true epileptic attack, while the succeeding stage, consisting of intermittent spasmodic contractions of the extremities, is comparatively longer. During this stage the entire body is seen to take part in alternate rhythmical contraction and relaxation. The child is perfectly unconscious, and may involuntarily pass both urine and fæces. In the course of a few minutes to half an hour, according to the gravity of the case, the spasms gradually subside, leaving the child in a soporous condition. It is not uncommon for several convulsions to occur in succession, as repeated convulsive seizures create a susceptibility from which the nervous system recalls itself with difficulty.

The *prognosis* depends upon the nature of the exciting cause and the course pursued by the seizure. When convulsions recur in rapid succession, or when associated with laryngismus stridulus, the prognosis becomes grave. Likewise in convulsions occurring with uræmia or with meningitis, extensive hæmorrhage, or other serious intra-cranial lesion, the prognosis is grave. Should the convulsive habit become firmly established the child is quite likely to develop idiopathic epilepsy.

**Diagnosis.**—The differentiation of symptomatic from idiopathic eclampsia rests upon a proper examination of the patient for evidence of disease elsewhere. Thus, with convulsions ushering in the infectious fevers, there are always the symptoms belonging to the stage of invasion of the particular fever in question. In uræmic convulsions the urine tells the tale. Those due to reflex irritation give evidence of such a



source of irritation, and a purely rachitic case becomes self-evident by inspecting the child closely. Intra-cranial disturbances are recognized by characteristic symptoms present before the convulsions have made their appearance.

Convulsions occurring shortly after birth are usually due to meningeal hæmorrhage. Unilateral spasms may occur from cortical hæmorrhage, as a result of whooping-cough, trauma or idiopathic origin.

*Epilepsy* is to be suspected when repeated convulsive seizures occur in children over three years of age, notwithstanding the absence of any source of reflex irritation or other cause to account for the attacks. Other symptoms, such as aura and the stigmata of degeneration, are usually ascertainable.

**Treatment.**—All exciting causes must be removed at once when this is possible, and the predisposing cause is to be overcome by attending to the child's general condition. Constitutional remedies and a properly-selected diet, together with plenty of fresh air and sunshine, are indispensable here. (See *Rickets*.)

As gastro-intestinal irritation plays such an important rôle in the precipitation of convulsive seizures, the stomach and bowels should at once be emptied when the attack is suspected to arise from this source.

During the seizure every article of clothing should be loosened. If the convulsion lasts for any considerable length of time a warm bath, together with cold applications to the head, is indicated. In long-continued or recurring convulsions a hot pack or a weak mustard pack is more practical.

The most frequently indicated remedies are *Belladonna*, *Cuprum*, *Cicuta*, *Ignatia* and *Magnesia phos.*, basing our prescription purely upon the occurrence of convulsions. But when the convulsion is purely symptomatic, the results of treatment will be more satisfactory if we direct our attention to the exciting cause instead of looking upon the convulsion as an independent disease.

*Acon.*—High fever ; high arterial tension ; great restlessness. Febrile cases.

*Bell.*—Convulsions, with flushed face ; dilated pupils ; cerebral congestion ; throbbing carotids ; pyrexia. Indicated in those cases ushering in the infectious fevers, in some reflex convulsions, and in convulsions occurring in the early stages of meningitis.

*Cuprum.*—Convulsions beginning in the fingers and toes, becoming general, with marked cyanosis. Spasm of the glottis is associated with these cases. Convulsions occurring during the eruptive fever when the rash disappears ; whooping-cough ; meningitis (*Cupr. acet.*).

*Cuprum ars.* is most valuable in uræmic convulsions.

*Cina.*—Reflex convulsions from irritation of the intestinal tract, whether due to worms or not. The spasmodic movements are often confined to the eyes and face, continued with irregular jerkings of the extremities. In this respect it is similar to *Chamomilla*, which presents many of the premonitory symptoms of eclampsia, the child being feverish, irritable, and suffering with intestinal colic or painful teething. In such cases *Chamomilla* will frequently ward off a convulsion.

*Cicuta.*—The convulsion comes on suddenly without premonitory signs. The stage of tonic spasm is well marked, and the child may remain rigid for a long time, only a few jerks of the extremities being noticed during the attack. It usually points to cerebral effusion.

*Ignatia.*—Convulsions in nervous subjects brought on by fright or peripheral irritation. The vascular excitement of *Belladonna* is not present in these cases, and the face is inclined to be pale instead of hot and flushed, as in the latter remedy.

*Magnesia phos.*—Idiopathic convulsions ; defective nutrition of the nervous system. (See *Epilepsy.*)

*Opium.*—Convulsions in cerebral hæmorrhage. There is trembling of the whole body ; purplish color of face ; stertorous breathing and sopor ; post-epileptic stupor.

*Veratrum viride*.—Great vascular excitement with high arterial tension; opisthotonos; eyes injected and staring; intense cerebral congestion.

Beside these remedies compare also *Apis*, *Calc. carb.*, *Gels.*, *Helleb.*, *Hyos.*, *Stram.*, *Ipecac.*, *Sulph.* and *Zincum*, and consult the article upon *Meningitis* and *Laryngismus Stridulus*.

#### EPILEPSY.

Idiopathic epilepsy is a condition in which recurring attacks of unconsciousness with convulsions (*grandmal*) or without convulsions (*petit mal*) are firmly established, ultimately leading to an impairment of the subject's mentality.

**Etiology.**—Heredity plays a most important rôle in the etiology of epilepsy, the tendency springing not only from the presence of epilepsy, but also of other nervous diseases, such as hysteria, neurasthenia and insanity in the family history. Parental syphilis and alcoholism are looked upon as causes. In some of my cases pronounced rachitic changes were present and the convulsions dated back to infancy. The majority of epileptics show in more or less degree the evidences of degeneracy, both physical and mental; indeed many are vicious and criminally inclined, while others show a low moral standard or deficient intelligence. Startling exceptions in the form of geniuses, of course, are observed. As to age, the period of puberty furnishes the majority of cases. It only rarely develops before the third year. Sachs (*The Nervous Diseases of Children*) is of the opinion that *hereditary* (idiopathic) epilepsy is not as common as is generally supposed, many cases being accepted as such because a former cerebral lesion or a traumatism to the head has been overlooked owing to the disappearance of the paralysis and other symptoms due to such a lesion, from which, however, the epilepsy dated. To this category belong those cases of epilepsy associated with infantile cerebral palsies and defective general development of the brain.

The *exciting cause* of the seizure is most often found in disturbances of the digestive tract. Acute indigestion, either through reflex irritation or auto-intoxication, will frequently precipitate an attack. Reflex irritation from phimosis, eye-strain, worms, etc., exerts a similar influence. Emotional excitement, excessive physical exertion, and poorly ventilated or crowded apartments are most disadvantageous to the epileptic. In several of my cases the first seizure developed after a slight traumatism, the psychic effect no doubt being more to blame than the accident itself.

A constant pathological lesion is not to be found. Judging from our knowledge of the physiology of the brain and the symptoms produced by irritation and organic disease of the cortex in the Rolandic area, it is reasonable to suppose that the pathologic condition must be located here. Indeed, a number of observers, notably Van Giesen and Bleuler, have demonstrated changes in the cortical cells and in the neuroglia. Lesions in the basal ganglia have also been described. These are probably in the nature of secondary changes. Onuf (*Jour. Amer. Med. Asso.*, April, 1905) reports the findings in sixteen carefully conducted autopsies. He discovered in ten cases marked thickening of the pia over the fronto-parietal lobe. There was also atrophy in the thalamic region. Whether these thalamic changes are directly connected with the seizure, or only part of a general pathologic condition of the brain, it is not possible to say.

**Symptomatology.**—An attack of *petit mal* is characterized by a momentary loss of consciousness, unaccompanied by convulsions or other nervous phenomena. In children it is often looked upon as mere absent-mindedness or a fainting spell; in older subjects it is more likely to be confounded with vertigo, with which, it is unnecessary to say, it has nothing in common. After this condition has once been fully established, a change in the child's mentality becomes manifest; it may go over into the convulsive form or exist in conjunction with the same.

Besides petit mal there are numerous other forms of incomplete seizure, all however attended by *momentary loss of consciousness*. There may be merely twitching of certain muscles, notably in the arm and face; a sudden impulse to run forward or perform other automatic movements, unconsciously. Sometimes *coma* exists without convulsions, or the child may have auræ for a long time before the convulsions make their appearance. There are also certain *psychic equivalents* of the epileptic seizure, in the form of maniacal and other insane acts. Following the fit, the patient may for several days perform acts for which he is irresponsible.

An attack of *grand mal* is very similar to an attack of infantile convulsions; but other conditions are added thereto, and the various stages are more sharply defined and characteristic. The following stages are to be observed:

(1) The *aura* or *prodromal* symptoms. This usually consists of a sensory disturbance, which may be variously described as a tingling; feeling of numbness; crawling; sensation of a gust of wind directed upon the affected part; hallucinations of sight, smell and hearing. There may also be motor disturbances, and the character of the aura will in many instances point to involvement of a special area of the cerebral cortex.

(2) The *initial cry*. This marks the commencement of the stage of tonic spasm. The patient utters a loud cry, as a result of the spasmodic contraction of the respiratory muscles forcing the air through the partially closed glottis, whereupon he falls to the ground as if shot. With this stage, unconsciousness also sets in.

(3) The *tonic spasm*. During this stage the body is perfectly rigid, the legs extended, the arms flexed and the hands clenched, the thumbs being pressed into the palms of the hands by the fingers. The head may be retracted, causing opisthotonos (young children), or it may be drawn to one side, the eyes being fixed and pointing in the same direction. The pupils are immovably dilated. The face, at first pale, now

becomes reddened, and even cyanotic, if the stage is prolonged. The jaws are set, and the tongue is frequently caught between the teeth. The stage of tonic spasm lasts for a period of about a minute, at the end of which time it gradually subsides, being followed by -

(4) The stage of *clonic spasm*. This consists of alternate relaxation and contraction of the muscles of the extremities and thorax, persisting for several minutes (seldom over five minutes). Through these movements the body is thrown into violent action, and frothy saliva is ejected from the mouth, the tongue quite frequently being caught between the teeth and badly bitten. Urine and fæces are frequently passed involuntarily. The movements gradually subside and the patient goes over into—

(5) The stage of *stupor*. Post-epileptic stupor is a profound sleep from which the patient may be temporarily aroused, but soon relapses into unconsciousness. This may last for several hours. The pupils are dilated.

**Prognosis.**—Cases of epilepsy coming under treatment early, providing there is no organic lesion or mental deficiency associated with the same, should not be despaired of as absolutely incurable. Traumatic epilepsy has been cured by early surgical interference. Hereditary cases offer a bad prognosis, as they usually present degenerative tendencies. "More favorable are those cases which come on during the period of dentition, or are caused by disturbances in the nutritive functions, such as chlorosis or anæmia; in fact, in all cases where it is possible to remove the cause" (C. G. R.). From this it will be seen why the best results in treatment are obtained by attending strictly to the patient's diet and hygienic surroundings, and by prescribing upon constitutional and general indications rather than upon the convulsive symptoms.

**Diagnosis.**—*Eclampsia*: Prior to the age of three years, longer duration, reflex irritation or organic disease ascertainable. The convulsions are liable to recur after a short time, while in epilepsy a long interval is usually present (BARTLETT).

*Hysteroid convulsions* are usually precipitated by emotional excitement; rigidity is marked, followed by irregular movements of the extremities; the duration is much longer than an epileptic seizure, and there is no biting of the tongue or involuntary micturition and defecation (GOWERS).

Other conditions to be thought of are *uræmic* and other *toxic convulsions*, and, in the case of *petit mal*, *syncope* and *vertigo* must be excluded.

Frequently we are called upon to make a diagnosis in a case where the occurrence of convulsions is not known. Thus, we may find a patient in post-epileptic coma, or have to deal with a case of nocturnal epilepsy where the convulsions have taken place unobserved. Post-epileptic coma is distinguished from uræmia by the pupils and the absence of albuminuria and casts in sufficient amount to indicate nephritis. The tongue should be carefully examined for scars. Strumpell lays stress upon a careful inspection of the conjunctivæ and face for punctate hæmorrhages. When these are seen in a patient who awakens in the morning dull and confused we have strong presumptive evidence of nocturnal epilepsy. When this is associated with enuresis the presumption is still stronger. Besides, in forming an estimate of the true nature of any condition associated with disturbed or temporary loss of consciousness the family history and the child's physical and mental development play an important rôle.

**Treatment.**—All sources of reflex irritation, such as phimosis, cicatrices, errors of refraction and nasal defects must be corrected at once. The diet is of great importance. The patient should be kept mainly on a vegetable diet, allowing milk regularly, and poultry and fish only occasionally; furthermore, the stomach must never be overloaded, and, besides prohibiting meat, all indigestible articles of food, such as pastry, rich desserts, etc., must be strictly avoided (BAILEY).

In cases of malnutrition, meat may occasionally be allowed.



but a liberal meat diet is always bad for children. Cases in which convulsions had ceased under an exclusive vegetable and milk diet invariably relapse when meat was allowed, no change in the medicinal treatment having been made (THOMPSON, *Practical Dietetics*). Codliver oil is indicated in the rachitic and strumous.

It is a noteworthy fact that an excess of *Indican* is found in the urine of many epileptics just about the time of the seizure (HERTER), being formed in the intestines from the excessive putrefaction of proteids. This points to the necessity of preventing intestinal putrefaction, which may be at least partially accomplished by careful regulation of the diet. Indicanuria is produced not only by the ingestion of albuminous food, but also as a result of muscular atony of the stomach and hypochlorhydria. As the lactic acid bacillus is antagonistic to the colon bacillus, and should in fact predominate over the latter in the small intestines in childhood, it is readily seen why a milk diet and the prohibiting of meat is so beneficial in epilepsy.

Excessive physical exertion must be avoided, while judicious out-of-door exercise proves of the greatest benefit.

During an attack the patient should be protected from doing himself injury. A towel or other available article may be inserted between the teeth to prevent biting the tongue, and the clothing should immediately be loosened. The inhalation of *Amyl nitrite* sometimes shortens the attack.

As before stated, the best results are obtained from remedies selected upon general indications, taking into consideration the patient's mental, temperamental and diathetic peculiarities; also, any disturbances in the alimentary, respiratory, genito-urinary tract, etc. For this reason such remedies as *Cicuta*, *Hydrocyanic acid*, *Uranthe crocata* and *Solanum* are rarely of positive value. On the other hand, *Argentum nitr.*, *Calc. carb.* and *phos.*, *Lycop.*, *Nux. om.*, *Pulsatilla*, *Silica* and *Sulphur* are of the greatest service. There are, however, a few anti-spasmodic remedies which are among the most use-

ful in epilepsy, but their action, as at once seen from a study of their pathogenesis, is a deep, selective one, not simply inducing transient functional disturbances. I refer mainly to *Cuprum* and *Magnesia phos.* *Magnesia phos.* has given me good results in cases of idiopathic epilepsy in so far that it greatly reduced the frequency of the paroxysms and lessened their severity; but as they were mostly dispensary and hospital cases, they were not observed for a sufficient length of time to judge of any permanent results. The prompt action obtained from this remedy in a case of *tetany* of long standing certainly points to its efficacy as an anti-spasmodic of great value.

Where disorders of the digestive tract and lithæmic symptoms are prominent conditions *Nux vom.*, *Lycopodium*, *Cina* and *Sepia* stand out prominently.

In *petit mal* I have obtained positive benefit from *Cannabis Indica*  $\theta$  in small doses. The seizures occurred less frequently and the child's general condition was improved. Another remedy from which I have obtained results and practically use as a routine in beginning the treatment of any case is *Santonin*. Whether or not worms are present, *Santonin* certainly is helpful in all cases of intestinal indigestion, and relieves many of the symptoms resulting therefrom.

The following *résumé* is given in order to call attention to the guiding indications for the important remedies:

*Arnica*.—Recent traumatic cases.

*Arg. nitr.*—Old-looking face, pupils dilated before paroxysm for a day or two, flatulent dyspepsia with cardiac palpitation, apprehensiveness and depression of spirits, attacks of hemicrania, periodic trembling of body and paralytic weakness, epilepsy from fright, masturbation, menstrual difficulties.

*Arsen.*—Anæmic, weakly subjects. Burning in the spine, burning in the stomach and bowels after eating, diarrhoea with smarting about anus. *Petit mal*.

*Bell.*—Violent convulsions, with marked cerebral convulsions; mania. Prodromal symptoms consist of flushing

he face, throbbing of the carotids; wild, staring expression; feeling of suffocation. During the interval, throbbing headache, vertigo, flushing of the face with burning heat, easily rightened, night terrors, enuresis. *Stramonium* is similar in many respects. Symptoms brought on by fright, with great nervous excitement; spasmodic constriction of the throat, gyratory movements of extremities and threatened convulsions. *Stramonium* is frequently of service when *Belladonna* has failed to give relief, or its chances for doing good have slipped by, as it is of no service in old cases. The cases in which *Hyoscyamus* has proven so beneficial are undoubtedly hysterical in nature, as Jahr intimates. Such causes as "disappointed love, jealousy and grief," mentioned under the etiology of *Hyoscyamus*, point to the hysterical element in these attacks, as also such symptoms as "attempts at swallowing fluids renew the attacks," and "inclined to talk a great deal after the attacks; slight wandering of the mind."

*Bufo*.—Bojanus (*Die Hom. Therapeutik in ihrer Anwendung auf die Operat. Chirurg.*, 1880) reported a series of twenty-two cured cases of epilepsy, among which four were cured by the use of *Bufo* alone, three with *Bufo* followed by *Salamander*, and two with *Bufo* in conjunction with *Lachesis* and *Ignatia*. He gives no special indications for this remedy. "After fright or onanism; attacks at night, followed by some hours of coma; loss of consciousness and falling down; tonic and clonic spasms; turgescence and distortion of face; bites tongue; involuntary emission of urine; the lower extremities are more in motion than the upper ones." —(C. G. R.)

*Calc. carb.*—Scrofulous diathesis and leucophlegmatic temperament. Anæmia; catarrhal and cutaneous affections; prominent belly; cold hands and cold, sweaty feet; sweating about the head. "Frequently indicated after *Sulphur*," or in conjunction with *Belladonna*.

*Cannabis Indica*.—Clinically useful in petit mal. Allen (*Handbook of Materia Medica*) gives the following symptoms

as marked: Absent-minded, *forgetful* of what he intended to write or speak so that he cannot finish a sentence; forgetful of his last words and ideas. *Unconsciousness* every few minutes. Misapprehensions concerning time and space.

*Causticum*.—Where the mind is affected and paralytic affections are associated with the epilepsy. Degenerative changes in the nervous system. Paralytic weakness after the seizure is marked. Cases caused by fright (also *Ignatia*, *Hyos.* and *Stram.*); nocturnal epilepsy (also *Cupr.*, *Calc. c.*, *Opium* and *Lycop.*), and those following suppression of eruptions (*Ars.*, *Cupr.*, *Calc. c.* and *Sulph.*).

*Cicuta*.—Violent epileptiform spasms, accompanied by puffed, bluish face; fixed, staring eyes; terminating in trembling and long-continued sopor. Intestinal irritation, with venous congestion of abdomen.

*Cimicifuga rac.*—Epilepsy associated with disturbances in the female generative organs.

*Cina* and *Santonin* are useful at some time or other in most cases. Ridding the intestinal tract of parasites is one of the first things to be thought of in epilepsy in children. Besides the symptoms directly referable to worms there are a number of others calling for *Cina*, particularly those referable to the disposition, the appetite and general nutrition.

*Cuprum*.—Clear, idiopathic cases without organic lesions. The attacks may have been precipitated by fright, mental excitement, or suppressed exanthemata. The attack is typical, and cyanosis is usually marked.

*Gels.*—Dull occipital headache before attack; languor; drooping of eyelids; easily frightened into diarrhœa; prolonged spasm of the glottis during attack.

*Glonoin.*—"Great congestion of head and right heart; during spasm he spreads his fingers and toes asunder."—(C. G. R.)

*Hypericum.*—After injury to the spine or peripheral nerves.

*Ignatia.*—This remedy is especially suited to ordinary

cases of epilepsy in children. They are exceedingly nervous and easily frightened, irritable and peevish, and difficult to control. Jahr considered it the most valuable remedy with which to begin a case.

*Indigo*.—Depression of spirits. Excitable, furious and easily angered before the attack. Melancholy, timid or gloomy after the attack (L. M. KENYON).

*Lachesis*.—Auto-intoxication marked. The stools are exceedingly offensive. Phlegmatic constitution with disposition to indolence and melancholy.

*Nux vom.*—Indigestion with attacks of canine hunger; constipation, tongue coated posteriorly, bad taste, headache on rising in morning, with irritability (*Lycop.*, great irritability after sleep) and anorexia, especially mornings. *Nux vom.* and *Lycop.* are very important general remedies for the epileptic.

*Opium*.—Prolonged post-epileptic stupor. Nocturnal cases, with mental derangements.

*Silica*.—Lack of animal heat; strumous and rachitic diathesis; neurasthenia; pale, transparent skin; profuse sweat after the seizure. "Epilepsy, the aura begins in the solar plexus. Chronic effects of fright and nervous shock. Great irritability; constant restlessness" (T. F. ALLEN).

*Sulphur*.—Scrofulous or psoric diathesis. It is unnecessary to describe the characteristic *Sulphur* child here. *Sulphur* is also important as an intercurrent, or in cases not responding to the usual list of remedies.

*The Bromides*.—When after careful study and conscientious efforts to control a case of epilepsy by means of a remedy chosen purely symptomatically in conjunction with faithfully carried out hygienic measures, the seizures still persist and recur with alarming frequency, whereby the patient's general condition and mental state are profoundly affected, then it becomes imperative to control the convulsions by physiological means. Not that the use of the *Bromides* is entirely unattended by unfavorable results, but these are compara-

tively mild and easily removed when compared to the undermining influence of oft-repeated convulsions upon the nervous system. Having a limited experience in the use of these remedies, I cannot do better than quote from the leading old-school authorities in neurology :

“ It is certain that very few cases have been permanently cured by the administration of *Bromides* ; but unquestionably they serve an admirable purpose in checking the number of attacks and in diminishing their severity. To accomplish this end the Bromide salts should be administered according to a definite plan. It has been my practice to give preference to the *Bromide of sodium*, which I employ, according to the age of the patient, in ten or fifteen-grain doses, three times a day. If given in a wineglassful of (alkaline) water after meals the gastric functions will not be seriously impaired. . . . In the case of nocturnal attacks the medicine should be given before going to bed [the entire daily dose], and at no other time.—(SACHS. *Nervous Diseases of Children*.) The method recommended by Seguin (*New York Med. Jour.*, March, 1890) has many followers. It consists in the administration of the larger part of the full daily dose shortly before the time when a seizure is to be expected. During the interval a much smaller dose is employed, and the bromide is always given highly diluted.

According to Bayley, the *Bromide of strontium* is less irritating, produces less acne and has seemed to him more satisfactory in results than those obtained from the *Sodium* or *Potassium salt*. He gives from ten to eighty drops of a saturated solution (each drop representing about  $\frac{1}{2}$  grain of the salt) after meals, well diluted. If favorable effect is noted, sufficient dosage is maintained to stave off the paroxysms. It has been claimed recently that the action of the bromides is augmented and that therefore the dose can be reduced if we entirely interdict the use of table salt at the time the patient is taking bromides.

As soon as the paroxysms are controlled the dose is de-

creased to a minimum, but the remedy should not be withdrawn immediately.

#### TETANY.

Tetany is a neurosis characterized by tonic spasms, occurring principally in the extremities. The spasm may remain confined to these parts or it may extend to other groups of muscles, thus involving the neck, trunk, thorax and abdomen. Strabismus and trismus may likewise be present, but they occur secondarily to the spasm of the extremities, contrary to the occurrence of trismus in lockjaw, where it is the primary manifestation. The spasm may be intermittent in character or persistent. In the majority of my cases it was persistent, recovery taking place in from one to two weeks.

**Etiology.**—The close association of tetany with laryngismus stridulus and rickets points to a general disturbance in the nutrition of the nervous system as the cause underlying these phenomena. Escherich has shown that in cases of laryngospasm, even in the absence of fully developed tetany, he could bring out some of its latent symptoms. The tetanic spasms can be explained upon the theory of an increased excitability of the gray matter of the brain, medulla and cord, resulting from the above-mentioned cause. It is not at all unlikely, however, that toxic irritation of the motor cells in the gray matter of the brain and cord produces the contractures, the toxin emanating from the intestinal tract. My own experience leads me to accept this explanation in view of the fact that I have seen tetany in infants without signs of rickets and in which the symptoms promptly disappeared after a regulation of the diet. An infectious origin is believed in by some. Koplik has seen most of his dispensary cases appearing in groups in the early spring months. The disease is not common in this country, and the majority of cases have been observed between the ages of one and five, males being more frequently affected than females. As exciting causes, intestinal irritation, persistent diarrhœa, teething, phimosis, ex-



posure to cold, and exhausting acute illnesses, such as typhoid fever and pneumonia, have been mentioned. Extirpation of the thyroid gland has frequently been followed by tetany in adults. Enlarged thymus is mentioned by Escherich.

**Symptomatology.**—Children who are old enough to express their feelings may complain of a tingling or numbness in the extremities preceding the attack. The spasm comes on suddenly, involving first the fingers and wrists, after which the toes and ankle-joints become fixed in a characteristic attitude. This is described as the “carpo-pedal spasm.” The fingers are straightened out and flexed at the metacarpo-phalangeal joints, while the wrists are likewise flexed and the thumb drawn in under the fingers. The feet are in a position of talipes equinus, with the toes extended and flexed at the metatarso-phalangeal joints. As stated above, the spasm may extend to other groups of muscles, causing opisthotonos, trismus, strabismus or dyspnœa, according to the locality affected. A cramp-like pain may be complained of in the muscles, and any attempt to extend the extremities or to place the child on its feet causes a painful strain upon them. After a variable period the spasm relaxes and an interval of several hours or days may occur. In particularly aggravated cases, however, the child does not appear entirely free from the spasmodic condition, evidence of slight rigidity and weakness of the affected muscles being present. There is another group of cases in which the hands and arms take the position assumed in driving horses (KOPLIK). The arms are pressed against the thorax and the thumbs are turned into the palms of the hand by the contracted fingers. Pressure upon the large nerve trunks and arteries of the extremities affected during the spasm will invariably bring on an attack. This sign is known as “*Trousseau's symptom*.” Another symptom obtained in tetany is known as *Chvostek's symptom*, and consists of a contraction of the muscles of the face after they have been tapped sharply.

Older children who have been walking and feeding them-

selves find it impossible to continue to do so. Even in the intervals between the spasms there is a tendency to have them recur from voluntary movements.

The *duration* has been variously given as from several weeks to several days. In this respect it is similar to chorea, many cases promptly recovering, while others practically show the choreic tendency or its after-effects throughout life. The *prognosis* is good, being favorable as far as danger to life is concerned unless the attacks are accompanied by spasm of the glottis or general convulsions, or pronounced spasm of the respiratory muscles. Under these circumstances a fatal outcome may result.

**Diagnosis.**—The carpo-pedal spasm of tetany is so characteristic that it cannot readily be confused with any other condition, especially when associated with laryngo-spasm. The other symptoms, indicating the increased irritability of the nerves, increased reflexes and Trousseau's and Chvostek's symptoms, serve to corroborate the diagnosis. Some authors go so far as to assert that laryngo-spasm is an incomplete form of tetany, as in many of these cases the latent symptoms of the disease, namely, Trousseau's and Chvostek's symptoms, can be elicited. When opisthotonos develops the matter becomes more complicated, but the absence of fever and disturbances of consciousness and the tendency to intermittency confirm the diagnosis of tetany.

**Treatment.**—The constitutional condition must be corrected by an appropriate diet and hygiene, that directed for rickets being especially suitable. In fresh cases in infants I begin by excluding milk for twenty-four hours, giving only barley water. Then, if the child has been previously fed on sterilized food I put it on pure, raw milk, properly modified to conform to the age and the state of the digestion.

The remedies most likely to do good are those exerting a direct influence upon the nutrition, especially upon that of the nervous system. According to Bartlett, there is no homœopathic literature upon the subject, and he recommends *Nux*

*vomica* and *Secale* as suggesting themselves symptomatically. A case of long standing coming under my care at the Children's Homœopathic Hospital was promptly relieved by *Magnesia phosphorica*, 3x trit., so that it was able to feed itself and walk. The cure could not be attributed to nursing alone, as it had come from other institutions. The child was dismissed cured, but the subsequent history could, unfortunately, not be ascertained. Since then I have given this remedy several times, but the duration of the disease under proper management is so short that it is difficult to say how much can be attributed to a medicine. As a constitutional remedy, and to avoid recurrences, I employ *Calcarca phos.*

#### AFFECTIONS WITH MOTOR DISTURBANCES: CHOREA.

Chorea, or St. Vitus' dance, is one of the commonest nervous diseases of childhood. It is a neurosis characterized by irregular, useless, involuntary muscular contractions in various parts of the body, usually of wide distribution, and associated with a loss of muscular tone and disturbed co-ordination of voluntary movements. The onset is acute and the course pursues a sub-acute character. The relationship of chorea to rheumatism is one of its most noteworthy features.

**Etiology.**—There are evidently two classes of chorea. In the one we can find no evidence of rheumatism or endocarditis, but must look upon the motor disturbances as arising from either malnutrition of the motor cells of the cortex or toxic irritation (possibly auto-intoxication), occurring in delicate and neurotic children usually as a result of prolonged indoor life and excessive pressure at school. For this reason we see so many cases developing in the spring of the year; in other words, toward the close of the school term. In every children's clinic the large number of pale, thin, ambitious children, mainly girls, that come regularly with symptoms of chorea in March and April stands in distinct contrast to the scarcity of these cases in the fall.

Hodge has shown that as a result of fatigue the nerve cells

shrink in size, their nuclei and nucleoli become shrivelled and the lenticular granules of the protoplasm, probably nutrient, disappear. While under ordinary conditions the cell is promptly restored to normal after a period of rest, a much longer time, and sometimes a protracted period of rest, is required for this restoration in anæmic, neurotic children.

Griesbach's interesting experiments with the esthesiometer have given valuable data in the study of school-fatigue in children. When this method of investigation shows in a given case that recuperation is sub-normal, we should accept the result as a danger-signal, for if fatigue is prolonged it becomes cumulative and then complete recuperation is impossible so long as the child is kept at school (LA FETRA).

In the other class of cases the etiologic factor is plainly rheumatism. In fact, the conditions may coexist or the chorea follow promptly upon an attack of articular rheumatism. Again, the frequent association of endocarditis with chorea—variously estimated by different authorities—points to the close relationship of the two affections. Indeed, Heubner (*Kinderheilkunde*, 1903) goes so far as to say that the etiology of chorea is closely related to all rheumatic poisons, not only to those producing acute articular rheumatism, but the affection may also develop on the ground of a gonorrhœal infection; likewise in the wake of scarlet fever and other infections in which arthritis at times occurs.

Streptococci have been isolated from the blood and nervous system in a few fatal cases of chorea by Westphal and by Wassermann; tonsillitis has also been observed to precede attacks of chorea, as in rheumatism.

The opponents of the rheumatic theory of the etiology of chorea have been misled to a certain extent by a failure to understand the clinical course pursued by rheumatism in the child. If we remember that rheumatic infection does not necessarily mean polyarthrititis, but that certain forms of sore throat; vague joint pains or pains in the muscles and tendons accompanied by fever; growing pains and primary en-

docarditis itself are all manifestations of acute rheumatism or rheumatic fever in childhood, we will decide that a much larger number of our cases of chorea are rheumatic. Heubner, in fact, makes the rather sweeping statement that chorea is the commonest form of rheumatism in childhood.

Chorea is also closely associated with a rheumatic family history. In the cases in which I was unable to ascertain definitely its symptoms of rheumatism in the child there was almost invariably a strong evidence of the disease in the parents or in other members of the family. From this it would seem that a common toxic agent exists which is capable of giving rise to choreic manifestations if it affects principally the cerebral cortex, and rheumatic manifestations if the articulations and serous membranes are attacked—an explanation advanced by Hirt and others. Indeed, we may observe both the manifestations of chorea and rheumatism to a marked degree in certain severe cases of rheumatic fever, and the appearance of choreic symptoms in such cases offers a grave prognosis, as they indicate a high degree of toxæmia.

A neuropathic family history is found in a large percentage of cases, and epilepsy, insanity or alcoholism in the parents are undoubtedly potent predisposing causes to chorea. In this respect sex also plays an important rôle, as girls are far more frequently affected than males. Fright is an exciting cause in many cases. No matter to which class the case may belong, this mental trauma acts as a precipitant of the symptoms.

The largest number of cases are seen between the ages of seven to twelve; before the fifth year it is quite rare, and after puberty it usually disappears spontaneously, although cases have been observed in adults. This must not, however, be confounded with Huntington's chorea, which is a hereditary disease developing between the thirtieth and fortieth year, and presenting a most unfavorable prognosis.

The *pathology* of chorea is still obscure. As the action of the toxins upon the cerebral hemispheres would in all prob-

bility excite only vascular and nutritional changes they are difficult to demonstrate. The frequency of unilateral disturbances early in the course of chorea, the cessation of symptoms during sleep, the blunting of the mental faculties and the occasional psychic disturbances observed, indicate that the gray matter of the cerebral cortex is pre-eminently affected. Organic changes in the structure of the brain may lead to the development of choreiform movements, especially lesions following a cortical hæmorrhage. The term "*post-hemiplegic chorea*" has been applied to these cases, but the movements are, more strictly speaking, athetoid in character, usually unilateral, not ceasing during sleep, and associated with rigidity and other evidences of organic disease.

The presence of capillary emboli in the brain (*corpora striata*) was seriously looked upon as the specific lesion in chorea, but this occurrence has been shown to be purely accidental, resulting from a complicating endocarditis, and by no means an essential element in the disease.

**Symptomatology.**—The onset may be sudden or gradual. A severe fright may be followed within the course of a few hours or a day by evidences of extreme restlessness and a disposition to jerk and twitch in various parts. Ludicrous grimaces may be executed, and the child is unable to remain seated quietly in one position for any length of time. The arms are thrown into continuous irregular action and the legs crossed or shifted from one place to another. Voluntary actions are executed with difficulty, being characterized by extreme awkwardness and futility of purpose; the extremities become weak, so that the child drops every article from its hands and stands in a relaxed, swaying position, readily stumbling or tiring out, while speech may become so indistinct and muffled from involvement of the tongue and muscles controlling the larynx (*laryngeal chorea*) as to render it most difficult of interpretation.

A more gradual result is seen in those cases resulting from overpressure at school, anæmia following acute illness, or any

other of the slower-acting exciting causes. The child will give indications of gradually-increasing restlessness and awkwardness, the first condition resulting from the occurrence of involuntary muscular contractions, while the latter indicates disturbed co-ordination from the association of involuntary muscular contractions with all voluntary efforts. These phenomena may begin in one extremity or as a unilateral affection, the first symptom being paralytic weakness. The entire body soon becomes involved, and the apparent paralysis may disappear or simply share in the general muscular debility. These cases are described as *paralytic chorea*, monoparesis being the most common type. Church (CHURCH and PETERSON, *Nervous and Mental Diseases*) is of the opinion that many of these cases really belong to the neuritides or to a myelitis, or are combinations of these with chorea.

The movements observed in the face are a twitching of the eyelids and distortion of the mouth. The tongue exhibits marked choreic twitchings in the majority of cases, even in such where movements of the extremities are slight. Sachs (*Nervous Diseases of Children*) places especial diagnostic value on the movements of the tongue and associated facial action in propulsion of this organ, describing these combined movements as the "facies" of chorea. The tongue movements are slow and coarse, and propulsion of the tongue is attended with unnecessarily wide opening of the mouth, raising of the eyelids and eyebrows, and catching of the tongue between the teeth through choreic movement of the masseters.

The head may be turned from side to side and the shoulders alternately raised and lowered. The hands are alternately flexed and extended at the wrist, and the arms are thrown about in an irregular and jerky manner in severe cases. Attempts to control these irregular movements or to perform voluntary acts only intensify them, and the child may become unable to feed itself or execute other coördinate acts. When the child's hand is taken between the hands of the examiner, the irregular muscular contractions are readily felt. By



Directing the child to perform some voluntary act, the movements are demonstrated to the eye. The legs may be so affected by the muscular weakness and incoördination as to render it necessary to put the child to bed. Although sleep may be so disturbed as to exhaust the child to the extreme, and the great restlessness render it necessary to protect the child against falling out of bed, still, in the majority of cases, the movements abate on lying down and disappear entirely during sleep. The latter symptom is pathognomonic of chorea, serving to distinguish it from other motor disturbances.

The *temperature* is normal in most cases; an elevation of several degrees should lead to a suspicion of rheumatism or endocarditis. The *heart* is affected in the majority of severe cases of chorea. The percentage of endocarditis reported by the various writers on the subject varies greatly. Heubner found a murmur in 53 per cent. of his cases, but this does not necessarily indicate that all had endocarditis. In hospital cases the percentage is highest, because the severer cases come to the hospital. Nevertheless, I have had a number of bad cases in my hospital practice in which there was no endocarditis. Out of forty cases personally observed during the last two years, ten had endocarditis. In two others a murmur was found, but it was considered purely functional. Of his series, fifteen gave a history of rheumatism. Osler examined one hundred and forty cases two years after an attack of chorea and found evidence of organic heart disease in seventy-two of these patients.

In older children mitral regurgitation is closely associated with chorea. Beside organic manifestations, a cardiac neurosis is also encountered, inducing a group of symptoms which disappear with the disease. Both arrhythmia and a systolic murmur may be present, simulating valvular disease; but the murmur varies from day to day in intensity, is not transmitted, the pulmonary second sound is not accentuated, and hypertrophy does not take place. The condition has been called

*cardiac chorea*, and is supposed to indicate irregular innervation of the papillary muscles.

The mental state of the child is one of irritability, mental lethargy with deficient memory and power of concentration, and it may even assume a maniacal type of disturbance. Although true mental derangement is rare, it is not unusual to observe a highly-exalted psychical state, especially with relapses or acute exacerbations in severe cases. The face becomes flushed; the eyes are brilliant and have a wild, staring expression; there may be alternate crying and laughing or simply crying out, and the general condition becomes greatly aggravated. With proper management such outbreaks are only of short duration, but they may become of serious import when associated with fever and progressive exhaustion, even terminating in coma and death. This constitutes the *choreic status*, which, however, is fortunately seldom encountered.

The *course* of chorea is quite variable. Although usually described as a self-limiting disease, it is, nevertheless, one which can be controlled to a marked degree by medication, whereby its course may be materially shortened and the symptoms greatly moderated. On the other hand, although complete recovery is the rule, there are numerous instances in which as high as a dozen relapses have been noted, or in which the child carries the evidences of chorea to adult life. The average duration can be placed at about from two to three months, always remembering the possibility of relapses, especially in girls. In a series of dispensary cases reported by Bayley (*Trans. Hom. Med. Soc. of Penn.*, 1896) the average duration from the time of onset was 19.4 weeks, and from the time of beginning treatment it was 12.1 weeks. In private practice the course is considerably shorter than these statistics indicate.

**Diagnosis.**—The main source of error in the diagnosis of chorea will arise from confusing it with the motor disturbances of such conditions as *post-hemiplegic chorea* and *athetosis*, which are postplegic movements associated with paralysis.

of cerebral origin, and those of *Freidreich's ataxia*, *multiple cerebro-spinal sclerosis* and *hysteria*.

The history of the case, the facies of chorea, the characteristic movements and the association of rheumatic symptoms on the one hand and the absence of signs of an organic nervous affection on the other should render the differentiation easy.

**Treatment.**—As soon as evidences of chorea are observed the child should be taken from school and every effort made to eliminate from its life all excitement and mental and physical strain. The child must be treated with patience and kindness. Parents should be impressed with the fact that it is utterly impossible for the child to control its movements, and that scolding or constantly calling the patient's attention to his condition will only aggravate the symptoms. Rest in bed is indicated in all grave cases or those of abrupt onset. Bartlett advises against all forms of physical exercise, but a sojourn in the country, with plenty of fresh air and out-of-door life, is of unquestionable benefit in cases of moderate severity. During convalescence I believe judiciously carried out exercises to be of great value.

The diet is of importance. Remembering the rheumatic element in these cases, fats, especially cod liver oil and butter, are of decided value. The free use of meats should be interdicted, but milk, eggs, cereals and vegetables may be taken liberally.

Extreme restlessness, insomnia and mental excitement call for a warm bath at bedtime. Hot milk is also a valuable adjuvant in these cases.

The remedies from which I have obtained the most positive results are *Belladonna*, *Causticum*, *Stramonium* and *Agaricus*. Bartlett recommends *Agaricin* in the second decimal trituration in all cases not presenting strong indications for any other remedy.

Where rheumatic symptoms are prominent, *Actea rac.*, *Rhus tox.* and *Sulphur* are frequently indicated and of value.

In the choreic states I have found *Bell.* and *Stramonium* of service.

*Arsenicum*, the chief remedy of the old school, administered in the form of Fowler's solution, and *Ferrum* are useful when anæmia and other conditions pointing to these remedies are prominent symptoms.

*Agaricus*.—Spasmodic, jerky movements of the extremities and frequent nictitation of the eyelids (*Hyos.*). Sensation of coldness and tingling in various parts; paralytic weakness of legs. The active principle of *Agaricus* is not *Agaricin* but *Muscarin*. Bayley speaks of it with praise. Personally my experience has been chiefly with *Agaricus* in the second and third decimal dilution and it has done well as a routine remedy in the milder, non-rheumatic cases.

*Bell.*—Great mental excitement; delirium approaching to a maniacal condition; the face is flushed and the eyes are brilliant and staring; there is great difficulty of speech, and a sensation of dryness and choking in the throat.

*Stramonium* should be given if *Bell.* does not promptly relieve these symptoms, and if there is an incessant throwing about of the arms and a highly frightened behavior of the child.

*Caust.*—Paralytic chorea with speech defect. The child stands in a limp, relaxed condition; it is hardly able to walk or dress or feed itself; the voice sounds thick and unintelligible, and the tongue is protruded with difficulty. In such cases *Causticum* may be considered well-nigh a specific. In the last years I have been able to verify repeatedly the value of *Causticum* when these symptoms are encountered.

*Cimicifuga*.—Rheumatic pains in the small joints; endocarditis; after suppression of menses.

*Cina.*—Helminthiasis or intestinal indigestion.

*Coccul.*—Right-sided chorea; face puffed and bluish; hands and feet look as if frozen; paralytic symptoms.—(C. G. R.).

*Hyos.*—Constant twitching of the eyelids; angular gyratory movements, with inco-ordination; misses what he reaches for; silly expression of face, smiling at everything he hears; chorea after debilitating fevers.

*Ignatia*.—Highly nervous temperament; easily frightened; starts at the slightest noise; irritable temperament. Mild cases, developing after fright.

*Mygale*.—Constant turning of the head to the right side, occasionally dropping it on the shoulder.

*Nux vom*.—Sensation of numbness in the affected parts; frontal headache, constipation, indigestion, irritability and lassitude.

*Pulsatilla*.—Chlorotic subjects; mild, tearful disposition; functional cardiac disturbances. Chorea developing at the time of puberty.

*Stramonium*.—Chorea developing after fright. The symptoms are usually severe, and may approach the choreic state. (See *Belladonna*.) The movements are pronounced, but there is not that degree of paresis calling for *Causticum*.

*Sulphur*.—Protracted cases with frequent relapses; rheumatic family history; after suppression of eruptions. Other constitutional remedies which may be called for upon purely diathetic indications are *Calc. carb.* and *Phos.*, *Mercurius*, *Phosphorus* and *Silicea*.

#### SPASMUS NUTANS; HEAD-NODDING WITH NYSTAGMUS.

The syndrome of rhythmic movements of the head associated with nystagmus is a peculiar condition occasionally encountered in rachitic and otherwise poorly nourished infants. Of late this phenomenon has attracted considerable attention among pediatricists, and a number of cases have been reported in the literature from time to time.

Nystagmus may be the only symptom, or it may be the first symptom, other nervous manifestations, namely, head-nodding and laryngismus stridulus developing later, as occurred in one of my cases. Blepharospasm may also be present (AMBERG), and associated movements in the extremities (AUSCH) and temporary loss of consciousness (HADDEN) have also been observed. As a rule, the movements cease during sleep.

The majority of cases occur in infants under one year. The early signs of rickets are usually present. There is no pathologic lesion, but most probably the symptoms are due to irritation or exhaustion of the nerve centres for the muscles governing these movements. Henoch has pointed out that the nuclei of the oculo-motorius and the nerves governing the movements of the neck are adjacent, and that, therefore, they are readily irritated simultaneously. In many cases there is no doubt as to the exciting cause, namely, keeping the child in a dark room with the eyes exposed to the bright light of a window, analogous to the etiology of miner's nystagmus. All my cases have occurred in dispensary patients from the poor, crowded districts.

The *prognosis* is favorable, as the symptoms depend partly upon the underlying malnutrition or auto-intoxication which may be present in the case. The *treatment* is purely symptomatic, and is to be conducted upon the lines as laid down in the discussion of rickets. In congenital nystagmus, of course, the outlook is different, but here the symptoms exist from birth. I have not been in the habit of prescribing for the nervous symptoms alone, but have obtained the best results when treating the underlying disturbance.

#### HYSTERIA.

Hysteria is a psychoneurosis combining cerebral insufficiency with certain disturbances of the sympathetic nervous system, "A state in which ideas control the body and produce morbid changes in its functions" (MOEBIUS). Almost any organic disease can be simulated by this peculiar nervous derangement, for which reason its recognition and proper understanding are of the highest clinical importance. Children are by no means exempt from hysteria, and sex bears no etiological relationship to the disorder. Although it may be encountered in early childhood, it is rare before the tenth year, and most prevalent at the period of puberty and adolescence. Heredity plays an important rôle, a neuropathic family history

ory being present in most cases. As exciting causes, emotional disturbances—especially fright, grief, jealousy, and minor traumatism in which the mental shock occurring at the time of the accident is entirely out of proportion to the injury sustained—are inseparably linked with hysteria. In the latter instance suggestion also enters into consideration, being one of the strongest influences in exciting as well as in removing hysterical phenomena. Reflex irritations, such as tight and adherent foreskin or adherent hood of the clitoris, have been mentioned as exciting causes. To these must also be added the baneful influence of improper training and discipline, bad habits and various debilitating illnesses.

**Symptomatology.**—In reciting the symptomatology of hysteria, the general attributes and characteristics of the disease will be outlined, after which the special symptoms and clinical types will be considered. The first are spoken of as the *stigmata*, while the latter are designated the *accidents* of hysteria.

**STIGMATA.**—The *mental condition* is characterized by diminished will power, loss of memory and lack of determination, and indecision. Impressionability and irritability characterize the temperament. These subjects are very susceptible to suggestions, and the mood vacillates between sadness and gayety, uncontrollable paroxysms of alternate laughing and crying being a frequent occurrence.

*Disturbances of sensibility* are encountered as complete or partial cutaneous anæsthesia, or hyperæsthesia in certain localities. It is usually found in parts which are paralyzed, hemiplegia with anæsthesia being strongly indicative of hysteria. Irregular islets of anæsthesia are likewise characteristic of hysteria. The area of anæsthesia does not correspond with the distribution of special nerve trunks or to the areas of sensation supplied by the different spinal segments, but seems to conform rather to the cortical representation of sensory areas. The mucous membranes may be anæsthetic and the special senses become perverted or abolished, leading



to disturbance of sight, hearing, etc., or sudden blindness or deafness. The throat may become anæsthetic, so that we can irritate the fauces without producing gagging. Likewise anæsthesia of the nose, conjunctiva, larynx, etc., is to be encountered. The reflexes are not disturbed, as they are in organic lesions associated with anæsthesia.

The *motor disturbances* to be observed are a general retardation of voluntary movements and muscular weakness and incoördination. This is explained by the presence of anæsthesia and loss of muscular sense and of the power of mental concentration.

To the milder forms of *spasmodic affections*, belong notably globus hystericus; hysterical cough, hiccough and glottic spasm; spasm confined to certain muscle groups, notably those of the neck.

The *diathesis of contracture* (CHARCOT) is a tendency to rigidities and contractures, which can be demonstrated by inducing a forcible flexion or extension in a limb, or by irritating the muscles by deep massage or by means of the faradic current.

ACCIDENTS.—To the *accidents* of hysteria belong certain transitory disturbances manifesting themselves as convulsive seizures (*grand attacks*; *hystero-epilepsy*), or as motor and sensory disturbances of major degree, closely simulating a variety of organic diseases.

*Grand attacks* belong to the rarer forms of hysteria in childhood; but as they bear a superficial resemblance to epilepsy, they will be considered in full. The attack is preceded by depression of spirits and a sensory aura, most commonly the *globus hystericus*. This is described by the patient as the sensation of a ball rising into the throat and is due to spasmodic contractions of the pharynx and œsophagus. A general tonic spasm, which persists for a few minutes, marks the first stage of the attack. The child lies stretched out, with the limbs extended and rigid, the fingers and toes being flexed. Slow, rigid movements of wider range executed by

he arms, and flexion and extension of the feet, may be observed during this stage. The jaws are tightly closed, and respiration is slow and irregular or entirely suspended. The face assumes a bloated appearance, and the veins of the neck are prominent and swollen.

The clonic stage is ushered in by short, jerky movements involving the face and extremities. These movements increase in severity, but do not assume the regular clonic type of epilepsy, being more irregular and of a struggling character. Respiration becomes interrupted and sobbing. Biting of the tongue is rare, as is also involuntary defecation and micturition. After the course of a few minutes the movements cease abruptly, and a period of resolution or repose sets in—a condition simulating sleep. This may end the attack, or be succeeded by the period of *clownism*, during which the patient becomes fixed in a variety of rigid postures. Extreme opisthotonos is a common position observed in hysteria. A phase of large movements now follows, in which the subject may cry out in fear or rage and strike or bite at those about him. Peculiar sounds are sometimes uttered resembling, for instance, the barking of a dog, and, when associated with the above symptoms, constitutes spurious hydrophobia.

The period of passional attitudes observed in adults is very rarely seen in children. The period of *delirium*, in which the child sobs and pleads in a pitiful manner, or expresses various hallucinations, often terminates the attack, after consciousness is restored.

*Motor accidents* occur as paralysis and contractures. They are usually of sudden onset, as the result of fright or injury; less commonly they develop gradually. Hysterical paralyses correspond in their general characteristics with cerebral paralyses—there is loss of will-power to move the affected member. The legs are more frequently affected than the arms. There may be monoplegia or paraplegia, which may be followed by contractures, or the palsies may alternate

with contracture. The paralyzed part is frequently anæsthetic, and the anæsthesia corresponds to the cortical distribution of sensation, not being confined to one or more nerve trunks, as in peripheral nerve and spinal affections.

In hemiplegia the face escapes, with the exception of the eye-muscles, which are at times affected. Anæsthesia is common, while in organic cerebral hemiplegia it is rare. Again, the contractures of hysteria partake more of the nature of spasmodic voluntary resistance, and atrophy never takes place excepting as a slight amount of wasting resulting from non-use. Loss of power is not absolute, and the degree of paralysis may vary from day to day. The gait also differs from that observed in cerebral palsies in that the leg is dragged along in a limp condition, not being swung out in a lateral direction, by which the foot is made to describe an arc.

Paraplegia may exist as a purely ideational palsy, rendering walking impossible, or there may coexist disordered function of the cord, indicated by increased knee-jerk, spurious ankle-clonus, retention of urine and spinal tenderness.

Contractures may exist independently or in association with paralysis and anæsthesia. The extremities are most frequently affected. When the hands and feet are affected, the fingers and toes are flexed. With involvement of the larger joints there is extension, so that the arm and leg are held out straight. Contractures may occur in monoplegic, hemiplegic or paraplegic distribution. In deep sleep the rigidity usually disappears.

*Astasia abasia* is a condition of lost co-ordination for walking and standing. It is produced by alternate contractions of antagonistic groups of muscles.

*Hysterical coxalgia* is a most important subject presenting itself for consideration to the pædiatrist. No doubt, the numerous cases of so-called reflex paralysis and coxalgia reported as having been cured by circumcision belong to this category. Apparently, every subjective and objective symptom of hip-joint disease has been mimicked by this neurosis,

and nothing short of a careful examination under an anæsthetic will serve to differentiate a pronounced case from true hip-joint disease. This holds good for other joint affections in which fixation and pain without any objective signs are present. In order to expel all doubt it may become necessary in an obscure case to resort to the tuberculin test.

*Tremors* and *rhythmical spasms*, the latter simulating chorea, are other motor accidents deserving mention.

*Sensory Accidents.*—A *pseudo-meningitis* is occasionally encountered, and is distinguished from true meningitis by the history of the case, the absence of slowing or irregularity of the pulse and active pupils. In other respects it bears a close similarity to meningitis, presenting intense headache; vomiting; fever; vasomotor streaks (*taches cérébrales*), and rigidity of the neck and extremities. Recovery, however, takes place, and a careful study of the patient reveals other evidences of hysteria.

*Spinal tenderness* may be confined to the region of a few vertebræ and closely simulate Pott's disease; but if the patient's attention can be detracted momentarily quite a considerable amount of pressure will be borne without causing pain.

*Visceral Accidents.*—Disturbances in the respiratory tract show themselves as *aphonia*, usually developing suddenly after a fright, the voice being lost, but cough persisting; *dyspnœa*, due to laryngeal or diaphragmatic spasm; *tachypnœa*, sudden attacks of extremely rapid breathing, presenting alarming symptoms, without the evidence of physical signs to account for the same, and *pulmonary congestion*. The latter is rare. It may produce cough with bloody expectoration, and simulate phthisis.

In the digestive tract, *vomiting*, *globus hystericus*, *œsophageal spasm*, *anorexia* and *obstinate* constipation are to be observed.

Frequent urination of large quantities of pale, limpid urine or complete anuria, sometimes retention of urine, are the disturbances encountered in the urinary tract.

*Vasomotor and Trophic Accidents.*—*Cutaneous hæmorrhages* and *gangrene* of the skin are among the rare hystero-neuroses, while *erythema* and *vesicular eruptions* are commonly met with. *Dermatographism* is occasionally observed.

*Muscular atrophy* and *fibro-tendinous contractures* are rarely well marked, although they may develop to a sufficient degree to require tenotomy in cases of long standing.

The muscles do not give the reaction of degeneration, although they may be partially atrophied and show a quantitative loss in electrical excitability.

The *prognosis* of hysteria is especially favorable in children, as they are readily influenced by suggestion, and, if the proper surroundings and intelligent treatment can be provided, recovery is generally comparatively rapid. The accidental disturbances, as a rule, disappear spontaneously after a variable period of months or years, or they may be purely transient. The mental state can, however, seldom be improved beyond a certain limit, and the hysterical temperament will persist throughout life in the majority of cases, even reflecting itself upon the offspring.

Sensory accidents are stubborn in their course, bringing considerable suffering to the patient and much anxiety to the friends and attendants. The spasmodic manifestations can usually be cured promptly if the patient can be taken from their parents and kept under intelligent care.

In the *diagnosis* much importance is to be attached to a recognition of the stigmata of hysteria; in other words, the hysterical temperament, in conjunction with the emotional origin of the ailment and the polymorphous and changeable character of the manifestations. Beside this, the differential features serving to separate hysterical from organic diseases, as pointed out in the symptomatology, should serve in leading to their recognition. This applies particularly to paralytic affections, which are of especial interest to the pædiatrists.

In coxalgia an anæsthetic may be required to remove any doubt in establishing the condition under consideration.

Hystero-epilepsy is rare in children, and its differentiation from epilepsy has been discussed in the article upon that subject.

**Treatment.**—The general management of hysteria resolves itself into removing all exciting causes, isolation being the most effectual method for this purpose; attending to the removing of all sources of reflex irritations, such as phimosis and errors of refraction, and building up the constitution by means of regular calisthenic exercises, a highly-nutritious diet and a liberal amount of sleep.

Suggestion presents itself as a most potent agent in restoring the patient's confidence and overcoming the various disturbances which have an imaginary origin. In managing cases of paralysis our main effort must be in the direction of promising the patient that the line of treatment employed will bring positive results. To emphasize this suggestion such adjuvants as massage and electricity are employed with benefit. This does not, however, apply to ill-managed cases of long standing, in which the surgeon's aid must be sought.

The beneficial results following upon even the most trivial surgical measures resorted to in hysterical subjects is a noteworthy clinical fact, which often can be taken advantage of as a justifiable means of treatment.

Medicinal treatment serves a twofold purpose, namely, by augmenting the force of the suggestions and also by improving the patient's general condition and correcting the various disturbances in the nervous system and other localities. It is needless to mention the close relationship existing between neurasthenia and hysteria in children, and, therefore, remedies which will improve the nutrition of the nervous system cannot fail to influence the hysteria. Such *remedies* as *Picric acid*, *Calcareo carb.*, *Silicea* and *Phosphorus* exert a potent influence in this direction.

Remedies possessing notably hysterical symptoms are *Ignatia*, *Hyoscyamus*, *Aconite*, *Asafœtida*, *Moschus* and *Valerian*. The efficacy of drugs in such conditions as hysterical palsy

and hystero-epilepsy is doubted by many. Arndt (*Practice of Medicine*) expresses the opinion that "they are often helpful, especially in times of great emotional excitement." If that were the case, a remedy should be useful as well at any other time when its symptoms are present, even if the disease be hysteria.

An unfortunate error often made in managing hysterical subjects is to look upon them as simply imagining their troubles and, therefore, requiring no treatment. Nowhere more than in hysteria does it require firm yet gentle supervision and persistent and encouraging suggestion to lift the patient out of his imaginary fears and afflictions. With a hysterical child we have a campaign of education before us which must be carried out up to the time of adolescence. The parents are often these children's worst enemies, and isolation is, therefore, one of the first steps in the treatment of a confirmed case.

#### PARALYTIC AFFECTIONS: CEREBRAL PALSIES.

The *cerebral palsies* of childhood comprise a group of conditions which may be either of intra-uterine onset, or which are acquired during parturition or at a still later period. Cases of intra-uterine origin are usually developmental in character, and to this group belong porencephalia, agenesis corticalis and other defects, although evidences of hæmorrhage and sclerotic changes, as a result of traumatism, foetal meningo-encephalitis and syphilis, have been observed in rare instances.

In birth-palsies, hæmorrhage is the primary lesion. It occurs frequently in protracted labors, and although forceful pressure may directly induce a hæmorrhage, still it does not play as important a rôle as long-continued compression of the head in the pelvic straits or within the uterus. It has also been supposed that undue pressure upon the trunk during the extraction of a breech presentation may be the direct cause for the rupture of a bloodvessel in the brain. The bleeding takes place from the capillaries and veinules of the pia mater or



choroid plexus in most cases, more rarely from the longitudinal sinus and veins, and almost never from an artery. Venous congestion attending compression of the cord and asphyxia may give rise to a pial hæmorrhage, but the weight of evidence is in favor of attributing the majority of cases of asphyxia neonatorum to hæmorrhage. A new-born infant therefore, with pallid asphyxia should be looked upon as most likely an apoplectic one unless good reasons for some other cause are at hand.

Where the amount of blood-extravasation is not sufficient to cause death, it ultimately is absorbed or becomes organized with consequent sclerosis of adjacent areas of brain-substance and developmental retardation. The symptoms attending such a condition will naturally depend on the locality affected.

The cerebral palsies encountered later in child-life are the result of either hæmorrhage, embolism or thrombosis. A cerebral abscess or tumor may likewise cause definite paralytic manifestations, but in their etiology and clinical course they differ distinctly from the foregoing conditions. Hæmorrhage at this period of life is more frequently meningeal than cerebral. It may result from traumatism, arteritis, or from a sudden and severe venous congestion of the brain occurring during a convulsion or during a paroxysm of whooping-cough. I have seen two cases resulting directly from whooping-cough. The convulsion is probably the result of the hæmorrhage, and not *vice versa*.

Birth-palsies are usually bilateral, that is, diplegic or paraplegic, while the later palsies are most frequently hemiplegic. Sometimes hemiplegia attacks an infant in apparently perfect health, the symptoms coming on with fever, followed by convulsions and hemiparalysis. Strumpell advanced the theory that these cases were infectious, and that an acute inflammatory process in the cortical gray matter of the motor area was the primary lesion. Pathologically and etiologically it was supposedly similar to poliomyelitis, for which reason he named it "*Acute Polioencephalitis of Infants*." Osler and Sachs

from a study of a number of these cases, question the correctness of this view, claiming that the lesions are probably always hæmorrhagic. In spite of the opinion of the high authorities just quoted, I believe with Mills, Holt and others that we do encounter cases whose mode of onset and clinical course certainly appear to bear out Strumpell's theory of an inflammatory lesion of infectious origin. Again, I believe that in some cases the symptoms are purely *toxic*, and we know that in adults apoplectiform attacks without hæmorrhage frequently occur as a phase of uræmia. In such cases we are surprised to find no gross lesion at the autopsy. Recently a colored child, two years old, previously perfectly healthy, was brought to my clinic for convulsions occurring daily and confined to the left side of the body. There was hemiplegia, although considerable improvement was manifest. The condition developed suddenly six weeks previous, the child being seized with these one-sided convulsions and temporary loss of consciousness, followed by hemiplegia. There was also fever continuing for several days. Why hæmorrhage should occur in such a case is hard to explain, but an encephalitis is quite conceivable.

Abscess is most frequently secondary to suppurating otitis media.

Sinus thrombosis results from extreme anæmia in conjunction with feeble heart's action occurring during exhausting illness, or from infection from the middle ear. In such cases thrombosis of one of the lateral sinuses, with its characteristic symptoms, results. Embolism is most frequently associated with endocarditis, only in rare instances originating from clots which have formed in the left auricle or elsewhere.

**Symptomatology.**—The lesions just enumerated may be productive of a variety of manifestations, for which reason we may encounter either *hemiplegia*, *diplegia*, *paraplegia* or *monoplegia* in these cases. The last two are rare, especially monoplegia, and paraplegia is frequently only apparent—a careful examination also revealing evidences of paralysis in the arms, together with mental deficiency.

The mental condition is impaired and the head is usually small or irregular of form. Epilepsy develops in about one-half of these cases, assuming the true degenerate type of the disease.

*Diplegic* cases are congenital, or result from injuries sustained during parturition. As above stated, the lower extremities are most markedly affected, and athetosis is a prominent symptom.

The case shown in the illustration, which is a very typical one, did not have athetoid movements. The child was mentally deficient, but there had been no convulsions. Lack of mental development can be traced back to the earliest period of infancy and on account of the spasticity of the legs they do not learn to walk until very late. The rigidity in both arms and legs varies in degree; when pronounced it reminds one of the resistance encountered in bending a piece of lead, for which reason it has been described as "lead-pipe rigidity." Together with this there is a crossing of the lower extremities due to adductor spasm and a tendency to equino-varus. The gait is, therefore, extremely difficult or impossible, and the hands are usually not well under control, being entirely helpless when athetosis is marked. A type of congenital diplegia resulting from defective development of the pyramidal tracts in the brain and cord, seen in underdeveloped or premature children, has been described by Little, of London (*Little's Disease*). They are not deficient in mind, and the spastic condition usually improves with the development of the nervous system.

Sachs (*New York Medical Jour.*, May, 1896) has reported a series of cases of congenital cerebral agenesis occurring as a family disease, in which amaurosis, progressive debility and a fatal termination are the clinical features. More or less diplegia, with spasticity, is usually present. A number of these cases is reported in the literature under the name of "*Amaurotic Family Idiocy*."

The *prognosis* is unfavorable in all cases, but especially in

the diplegic forms, in which little can be done aside from improving the child's general condition by means of massage and faradism, or by surgical measures when necessary. The proper training of such cases is, however, of the greatest importance, through which means both the mind and body may be most wonderfully improved.

In recent cases of hemiplegia the child must be dealt with purely symptomatically, and remedies are of decided use



FIG. 39. A CASE OF CEREBRAL DIPLEGIA IN A CHILD TWO AND ONE-HALF YEARS OLD, SHOWING SPASTIC RIGIDITY OF ARMS AND LEGS.

here. Massage and faradization of the extensor muscles, and mechanical contrivances to overcome contractures, are generally useful later on. *Arnica*, *Kali hydrojod* and *Sulphur* are aids in absorbing the hæmorrhagic extravasations, while *Caustium*, *Cocculus* and *Cuprum* frequently exert a beneficial influence upon the paralytic symptoms.

## ACUTE ANTERIOR POLIOMYELITIS; INFANTILE SPINAL PARALYSIS.

Poliomyelitis is perhaps the commonest form of paralysis encountered in childhood and, as the name implies, is an inflammatory infection of the spinal cord, the lesion being a focal one and practically confined to the gray matter constituting the anterior horns. The *acute form* is almost exclusively a disease of childhood, being most frequently encountered in the later period of infancy. Poliomyelitis is occasionally encountered in adults, and then usually assumes a *subacute* type.

Regarding the *etiology* nothing definite is known, but, judging from the clinical course of the disease, namely, rapid onset, with fever and other constitutional disturbances, its great predilection for the age of childhood, and the frequency of endemic and epidemic outbreaks—we are justified, in the present state of our knowledge, in classing it among the infectious diseases.

**Pathology.**—The site of predilection is the cervical or lumbar enlargement of the cord and the inflammatory process extends into the anterior horns by way of the median branches of the ventral spinal artery. The toxine responsible for the lesions excites either a focal hæmorrhagic myelitis or the initial lesion may be embolism or thrombosis of one of these arteries (MARIE). The multipolar cells of the anterior horn undergo atrophy and the inevitable result is paralysis and atrophy of the muscular fibres supplied by these nerve cells. In old cases the horn appears shrunken in size and inflammatory tissue occupies the place of the multipolar cells.

**Symptomatology.**—The onset is rapid, with fever of a moderate degree and some constitutional disturbances, even delirium and convulsions having been observed, or the symptoms are so slight as to escape notice, and the child develops an extensive paralysis, without any apparent cause for the same being ascertainable. The stage of invasion, therefore,

varies from a few hours to several days, and is of little diagnostic value.

The paralysis is usually of extensive distribution in the beginning ; but as improvement sets in the paralysis becomes limited to those regions which have been most seriously affected, in which muscular atrophy also develops. With the onset of paralysis local tenderness in the limbs may be noted. The paralysis rapidly increases, remains stationary for a period of a week or two, after which it rapidly improves in certain regions, while in others prominent disability remains and wasting of the muscles sets in.

The reflexes are lessened or abolished, according to the extent of the paralysis, but control over the sphincters is rarely lost, such an occurrence indicating a grave outlook. The alteration in the electrical reaction of the muscles manifests itself as the reaction of degeneration. The growth of bones may be greatly retarded through involvement of their trophic centres in the cord.

The distribution of the permanent paralysis varies greatly in individual cases. It may involve one or more extremities or remain confined to a few muscles of an extremity. The lower extremities are most frequently affected, but seldom equally. In the leg the most common deformity encountered is *talipes equinus* with flexed leg, resulting from wasting of the extensor muscles. In the upper extremities the deltoid, the extensors of the wrists and the interossei are most frequently affected.

The *prognosis* is unfavorable in so far as recovery of function in the paralyzed and atrophied parts is concerned, although there is seldom danger to life. Reaction to faradic stimulation is always a favorable sign, even when the muscles have failed to respond to this test earlier in the disease. Early loss of the same, however, indicates a permanent paralysis in most instances.

**Diagnosis.**—The diagnosis of both the early as well as the late manifestations of poliomyelitis anterior may be beset

th difficulty. Until paralysis is well developed, the disease cannot be positively recognized, and then it may be confounded with *rachitic pseudo-paralysis*, *multiple neuritis* and *cerebral palsy*. The latter is of abrupt onset, is ushered in by a convulsion, and the paralysis is one-sided and uniform. In *multiple neuritis*, the reflexes are increased in a central lesion while in *poliomyelitis* they are abolished. In *neuritis* the onset is more gradual as a rule and there is pain, together with perching tenderness, along the nerve trunks. There is never any atrophy seen in *poliomyelitis*. Epidemics have been observed in which there were cases of peripheral as well as spinal paralysis. The pronounced muscular atrophy of *poliomyelitis*, therefore, together with the reaction of degeneration and the history, are the features of *poliomyelitis* by which it can be differentiated from other forms of paralysis.

**The Idiopathic Muscular Dystrophies** bear a close outward resemblance to the late manifestations of anterior poliomyelitis. They have been divided into a variety of clinical types, but are all closely related both etiologically and pathologically. The main point of distinction between these myopathies and *poliomyelitis* is their slow and progressive development, the symmetrical distribution of the atrophic changes, and the strong hereditary element and developmental factor in their etiology.

The *pathological changes* observed in progressive muscular atrophy take place primarily in the muscles themselves, and the various clinical types of the disease really come under one and the same heading from the pathological standpoint (ERB). The muscle-fibres at first become hypertrophied, undergoing subsequent atrophy. The connective tissue is slightly increased. In isolated cases degenerative changes have been observed in the cells of the anterior horn (chronic poliomyelitis).

The following types have been described:

*The Juvenile Type of Erb.*—In this form the muscles of the arms and shoulders are mainly affected.



*The Facio-scapulo-humeral Type of Landouzy-Déjérine* (Infantile Form of Duchenne), in which the face, together with the arms and shoulders, are affected.

*The Peroneal Type of Charcot and Marie*, in which the peroneal muscles become atrophied. This may be followed

by atrophic changes involving the legs, trunk and upper extremities, and there is evidence of cord-lesions associated with the atrophy, showing itself as fibrillary twitching and reaction of degeneration.

*Pseudo-hypertrophic Paralysis* is a disease of early childhood, most frequently seen in boys, characterized by enlargement of the calves and buttocks, associated with atrophic changes. The muscles finally shrink, presenting the same condition as the other forms of atrophy. The characteristic symptoms produced are a waddling gait; difficulty of climbing up stairs and great awkwardness; enlargement of the legs and buttocks; lordosis; inability to arise from the ground without the aid

FIG. 50.—CLIMBING UP THE THIGHS IN PSEUDO-HYPERTROPHIC PARALYSIS (GOWERS). FROM BARTLETT'S DIAGNOSIS.

of the hands. In order to attain the erect position the child supports the hands on the anterior surface of the thighs and gradually pushes himself upright (Fig. 50).

**Treatment.**—The child should be disturbed as little as possible, not interfering with ill-judged applications of farada-

dism to the affected limbs during the acute stage, but wrapping them in cotton and enjoining absolute rest.—(BARTLETT.)

Later in the disease electricity proves of decided benefit. If the muscles do not respond to the faradic current the galvanic should be employed. The object is to produce muscular contractions in order to improve the nutrition of the muscle and restore function as far as that is possible. Passive movements and massage should be added to the treatment in order to overcome deformities. When once established, these will require surgical measures to correct them. The disability in a joint resulting from atrophy of one of the muscles either flexing or extending the same is often satisfactorily corrected by a properly adjusted brace, which not only supports the joint but also prevents deformity.

The remedies indicated in the early stages are such as will control the inflammatory condition, with the hope of lessening the secondary destructive changes. *Acon.*, *Bell.*, *Bry.*, *Gels.*, and *Rhus tox.* should be studied and carefully differentiated if there is a sufficiency of symptoms to prescribe upon. Otherwise, *Bell.* should be given the preference. *Mercurius* may be given with a view of absorbing exudation as promptly as possible. *Plumbum* is indicated at a later period. "The symptoms of chronic lead-poisoning correspond very closely with the symptoms of poliomyelitis."—(C. G. R.) It has seemed to me that the administration of *Causticum* has in some cases at least improved the tone of the muscles after the condition had come to the point of standstill.

#### FAMILY ATAXIA.

Family ataxia, also known as *Friedreich's disease*, occurs as a family disease, several or all of the children of a family being attacked by a degenerative process of the posterior and lateral columns of the spinal cord as a result of teratological defects in its structure (neuroglial sclerosis). The first symptoms usually make their appearance shortly before puberty, a period at which the processes of growth and nutrition are

taxed to their utmost. When there are successive in a family they usually develop at a progressively increasing earlier period of life. An acute infectious fever may also hasten the development of symptoms, leading to its occurrence in early childhood.

*Hereditary cerebellar ataxia* of Marie is characterized by a similar defective condition involving the cerebellum; but it develops after puberty, and is accompanied by pronounced choreiform movements, increased deep reflexes, and optic-nerve atrophy, symptoms not belonging to spinal ataxia.

**Symptomatology.**—One of the earliest symptoms noticed is an awkwardness in the legs, marking the beginning of the ataxia. Later the arms become involved. There is first unsteadiness in walking and standing, the child sways from side to side in attempting to maintain its equilibrium. As the muscular sense is not lost, the condition depending entirely upon incoördination, no increased difficulty in standing is noticed when the eyes are closed. The ataxia is associated with gradually increasing loss of power. The knee-jerk is lost early in the disease. This distinguishes it from the cerebellar variety, in which there is also at times an ankle clonus.

Disturbances of speech develop as incoördination becomes general. The speech is irregular and jerky, and lacks modulation and rhythm.

Nystagmus may develop later in the disease, being especially noticed with lateral rotation of the eyes. The expression is one of apathy and indifference, although the intelligence is not impaired early, but it becomes more or less retarded with the progress of the case, as does also the physical development. Shortening of the foot, with exaggerated plantar arch and retraction of the great toe (*club-foot* and *hammer-toe*), is a common deformity of family ataxia. Another deformity is dorso-lumbar scoliosis. These deformities may develop before ataxia becomes pronounced, and constitute an early sign of the disease.

The *course* is that of a progressively-increasing and hopeless malady, but remissions or aggravations may take place. There is nothing in the disease itself to cause death, for which reason the person so afflicted may live to adult life.

Isolated cases are to be differentiated from *cerebellar ataxia*, *chorea* and *multiple (insular) sclerosis*. In the latter there is characteristically scanning speech, spastic gait and intention tremor.

#### HEREDITARY SPASTIC PARAPLEGIA.

This is a rare disease, first described by Strümpell, which develops in early childhood and pursues a progressive course. The pathologic findings are degeneration of the lower part of the pyramidal tracts. There is no cerebral involvement, consequently no history of birth injury or convulsions during infancy followed by paralysis, and the patients do not show evidence of mental deficiency nor do they become epileptic, as in the cerebral diplegias and paraplegias resulting from hæmorrhage. Although the symptoms may not develop until adult life, still there seems to be no doubt that it is a teratological defect in the upper motor neuron. The *symptoms* are marked by spasticity and hypertonus of the muscles of the lower extremities, without sensory disturbances or involvement of the sphincters. The reflexes are increased. Bayley (*Jour. Nerv. and Ment. Diseases*, Nov., 1897) reported a series of cases in which the disease was traced back through five generations. The pathological findings are those of a degenerative process in the pyramidal tracts, the direct cerebellar tract and the columns of Goll.

The course is slow and progressive.

#### SYRINGOMYELIA.

Syringomyelia is a disease of the spinal cord in which the spinal canal becomes pathologically enlarged as a result of gliomatous infiltration, which subsequently breaks down. By the same process new canals of considerable length

may be formed within the gray matter of the cord. Although a rare disease in childhood, still it has occasionally been encountered in young subjects. As to its etiology nothing definite is known, excepting that embryonal neuroglial tissue degenerates or becomes the seat of hæmorrhage.

The symptoms resulting from a central myelitis or from a hæmorrhage into the cord—the latter, at times, occurring during parturition—cannot be distinguished from those belonging to glioma.

**Symptomatology.**—The disturbances of syringomelia may be divided into several groups. Involvement of the sensory pathway in the gray commissure and posterior horns and columns gives rise to loss of pain and heat perception, without, however, loss of the tactile sense. This anæsthesia may be so complete and extensive as to render the patient insensible to almost any kind of pain and expose him to many dangers.

Motor disturbances develop later than the sensory, and present paralysis of groups of muscles of a limb, usually becoming bilateral and accompanied by trophic changes. The reaction of degeneration is present. These symptoms indicate involvement of the anterior horns and pyramidal tracts.

Vasomotor disturbances, cyanosis, coldness, cutaneous eruptions and dermatographia may accompany the above process. Trophic changes, with resulting atrophy, fragility of bones, enlargement of the hands, and tendency to the development of whitlow and abscesses, are also to be noted.

The *course* is progressive, and results fatally when bulbar crises set in. In the *diagnosis*, the *idiopathic muscular dystrophies*, *hysteria* and *multiple neuritis* are to be excluded. The distinct features of syringomyelia are its gradual development and insidious onset, and the dissociation of touch and pain in conjunction with motor, trophic and vasomotor disturbances.

## MULTIPLE CEREBRO-SPINAL SCLEROSIS.

Multiple or *disseminated* sclerosis, as the name implies, is a degenerative process affecting the brain and cord as an irregularly scattered sclerotic process. The islets of sclerosis are found principally in the centrum ovale, crus, pons and medulla in the brain, and in the cord they are irregularly scattered, as a rule attacking the white matter more prominently than the gray. It is most common between the ages of twenty and thirty, but it may occur in children or even be congenital.

The *cause* of multiple sclerosis is probably to be found in an infection, but, judging from the numerous and often mixed infections noted, it seems unlikely that we have to deal with a specific organism.—(CHURCH.)

**Symptomatology.**—Owing to the widely-distributed lesions of multiple sclerosis a variety of disturbances are encountered in the nervous system. The characteristic and most prominent features of the disease are :

(a). *Motor.*—A coarse, jerky incoördination, especially in the arms, observed on attempts at voluntary movements. This intention tremor is associated with progressively increasing loss of power. The gait is spastic and is associated with deranged equilibrium (cerebello-spasmodic gait).

(b.) *Sensory* disturbances are practically confined to the eye. Nystagmus is a frequent symptom, and optic neuritis and atrophy may develop.

(c.) *Cerebral Disturbances.*—The speech defect, known as “scanning speech,” in which there is an undue separation and accentuation of the syllables of words, and a state of indifference, loss of memory and dejection, are the prominent cerebral features of the disease. A predisposition to hysteria seems to exist, and it is not uncommon to find hysterical manifestations complicating multiple sclerosis.

(d.) The deep *reflexes* are exaggerated, as a rule, but there may be a loss of knee-jerk, and paralysis of cranial nerves in some cases.

The *course* of multiple sclerosis is quite irregular. It may begin gradually and increase in a progressive manner, or it may begin abruptly as an apoplectiform attack, or with vertigo or visual disturbances. Remissions are not infrequent, and may lead to a belief that the disease has been checked; but complete recovery must be very rare, although Church considers it possible.

**Diagnosis.**—Multiple sclerosis is to be differentiated from *infantile cerebral palsy*, *hysteria* and *family ataxia*. In *infantile cerebral palsy* the history of traumatism during birth and the early appearance of diplegia, followed by mental retardation, rigidity and athetosis, will serve as a distinguishing feature. In *hysteria* the mental stigmata, the absence of nystagmus, and the presence of sensory disturbances and muscular rigidity, are of great significance, although both diseases may be associated in the same patient. In *family ataxia* there is inco-ordination and spasmodic muscular action; the knee-jerks are abolished, the muscles are flaccid, and the eyes are seldom affected, except by a slight degree of nystagmus, with lateral rotation of the eyes.

The *treatment* of these cases is very unsatisfactory. According to Arndt, *Arsenicum* is of especial value. *Tarantula* has also been recommended. Bartlett refers to the salts of *gold*, *lead* and *mercury*.

#### MULTIPLE NEURITIS.

Inflammation of several nerves occurring at the same time or in quick succession occurs mainly from diphtheria during childhood. Malaria, typhoid fever, scarlet fever, measles, influenza and acute rheumatism are responsible for some cases, but to a much less degree than the first mentioned infection. In marantic conditions and as a result of the cachexia of tuberculosis it may be encountered. Toxic cases, notably those seen in adults resulting from alcohol, arsenic and mercury are rare in childhood. There is a class of *idiopathic* cases that is quite puzzling. To this belong the rheumatic



cases following exposure to cold or resulting from over-exertion and those coming on suddenly with febrile symptoms, in the midst of apparently perfect health. Clinically they resemble acute poliomyelitis closely, especially when occurring in epidemics.

The lesions are a degenerative process in the axis-cylinders, not, however, affecting the nerve trunk uniformly and completely. This is associated with hyperæmia of the peri- and endoneurium. In some of the severer cases of diphtheritic paralysis degenerative lesions have been demonstrated in the cord and even in the brain in association with the neuritis.

**Symptomatology.**—The clinical course of diphtheritic paralysis has been described under *Diphtheria*. In non-diphtheritic cases there is first noticed a general weakness of the muscles, together with pain and tenderness along the affected nerves. Tingling and formication are also frequently complained of. The paralysis which results is usually of wide distribution, producing foot-drop and wrist-drop, inability to walk, and spinal curvature. Partial anæsthesia likewise develops, and considerable atrophy of the paralyzed muscles takes place. The knee-jerk is abolished, and if power of locomotion is not entirely lost the child shows marked ataxia in walking and standing. In the course of a few weeks improvement sets in, and after a time complete recovery is the rule, although permanent loss of function may persist. Permanent disability is rare in children and the prognosis is good, as the etiologic factors responsible for the unfavorable outcome in adults—such as alcohol—do not enter here. A fatal termination may take place in diphtheritic paralysis, or in other cases of rapid onset and wide distribution, in which the respiratory and cardiac innervation becomes involved.

**Diagnosis.**—The gradual onset, usually developing during the period of convalescence from an infectious disease or after exposure to damp and cold (rheumatic cases), the symmetrical distribution, and the accompanying sensory disturb-

ances, will serve to differentiate multiple neuritis from *poliomyelitis anterior*, as well as from the various *ataxias*. Its tendency to progressive improvement and recovery is another feature of diagnostic importance. The presence of pain is an important symptom, especially tenderness along the nerve trunks. In children it is often difficult to estimate the degree of pain, but, as Koplik says, the children resent being handled, and they cry most of the time and are restless at night, consequently it is safe to infer that they have pain. It is true, there may be pain in the early stages of acute poliomyelitis, but the general paralysis clears up more quickly and atrophy in a single limb or rarely in a portion of two limbs rapidly sets in. The electrical reactions are more typical and constant than in neuritis.

**Treatment.**—The child should be kept in bed and put on a plain, highly nutritious diet. Mild galvanization of the affected nerves and, as atrophy sets in, massage of the muscles are of great benefit. To overcome deformity in the extremities it may be necessary to resort to mechanical devices.

*Aconite*.—Recent cases following exposure. Tingling and formication in the affected parts is its chief indication. This and *Rhus tox.* are the chief remedies in idiopathic neuritis.

*Arsen.*—Malarial or cachetic cases; burning pains; general prostration. Marantic origin; cachexia.

*Argentum nitr.*—Ataxic symptoms.

*Causticum* is a most useful remedy for localized paralyses due to neuritis, or for the later changes of multiple neuritis.

*Gelsemium* is useful in the early period of infectious cases, notably in diphtheritic paralysis.

*Rhus tox.* is of great value in rheumatic cases. Traumatic cases call for *Arnica* and *Hypericum*, especially the latter.

#### SYMPTOMATIC AFFECTIONS: NEURALGIA.

Neuralgic pains may be observed in malnutrition and anæmia, particularly in chlorosis, or they may indicate a malarial infection. Hysteria is another prominent factor in

the etiology of various painful affections of childhood in which structural changes cannot be demonstrated, but on the whole neuralgia is uncommon before the period of puberty.

*Gastralgia* is a form of neuralgia which is usually the result of indigestion. It is discussed under the diseases of the stomach. Local irritations, especially carious teeth, are common causes for neuralgia. *Referred pains* have their special significance, *e. g.*, pain in the knee in hip-joint disease; abdominal pain in pleurisy; the various forms of headache resulting from eye-strain, nose and ear disease, etc.

Before a **diagnosis** of neuralgia can be positively made, it is essential to exclude all inflammatory conditions or sources of local irritation which might possibly cause the pains complained of. This is especially necessary in children, as serious organic disease may be overlooked by neglect of this precaution.

The **treatment** is mainly constitutional. A sufficiency of out-of-door exercise should be combined with a diet consisting especially of fats, milk, and vegetables. If anæmia is a pronounced feature, this should be corrected (see "*Anæmia*."). The most useful *remedies* in neuralgia are *Aconite*, *Arsenicum*, *Belladonna*, *Chamomilla*, *China*, *Colocynthis*, *Gelsemium*, *Rhus tox.* and *Spigelia*. The characteristics of these remedies must be taken into consideration in prescribing for a neuralgic affection, noting the locality, the character of the pain and the aggravation and amelioration. In chronic cases, constitutional treatment gives the best results.

#### HEADACHE.

A variety of conditions—notably anæmia, lithæmia, eye-strain, neurasthenia, hysteria and gastric derangements—give rise to headache as a symptom meriting special attention. In inflammatory and organic brain affections it is a prominent symptom, and in the infectious fevers and in uræmia it is quite constantly present.

*Migraine* is an essential headache, coming paroxysmally and resulting from nervous discharges in the cortical sensory centres. The *exciting causes* may be any of the disturbances capable of producing headache, such as mental or physical fatigue, eye-strain, acute indigestion, etc. The condition itself is usually hereditary, and is one of the manifestations of a neuropathic constitution, being, so to speak, a sensory epilepsy.

The *symptoms* of migraine in childhood are the same as those observed in adults, with the exception that they are not quite so severe and usually of less frequent occurrence. Scintillating scotomata are often observed, being described as fiery flashes or figures before the eyes. The pain may be confined to one side of the head, and is accompanied by nausea and vomiting, the latter giving relief, as a rule, although indigestion has nothing to do with these attacks excepting that it may act as an exciting cause. Other disturbances—*e. g.*, amblyopia; hemianopsia; aphasia; numbness and tingling in various parts of the body, followed by anæsthesia, and possibly paralysis—may be observed during an attack.

The *diagnosis* of migraine is based upon the paroxysmal nature of the attacks, the presence of nausea and vomiting without gastric derangement, and the accompanying sensory disturbances. *Symptomatic headaches* are recognized by their transitory nature and the presence of one of the causes enumerated above as causing the same. It is important both from the standpoint of prognosis and treatment to exclude *intracranial disease* in these cases by carefully searching for evidences of the same, and observing the patient over a sufficient length of time to determine the true nature of the case.

**Treatment.**—Children subject to migraine should be carefully dieted, especially avoiding sugar and starch, as these patients are usually lithæmic. Errors of refraction must receive prompt attention. One of the following remedies will usually be indicated during the paroxysm

*Arg. nitr.*.—Deep-seated pains in the temples of a boring or pressing character, relieved by pressure. Dimness of vision with vertigo, tendency to fall to the side. At its height there is trembling of the whole body and intense nausea. Attack preceded by chilliness, indisposition and loss of appetite.

*Bell.*.—Congestive headache; throbbing of the carotids; throbbing pains in the temples; face flushed. Often right-sided. The pain is worse lying down, and is temporarily relieved from sitting up.

*Cham.*.—Beginning with flickering and fiery zigzags before the eyes. Great irritability of temper.

*Glou.*.—Violent pulsations in brain from below upward; there is high arterial tension; vertigo; ringing in the ears and palpitation of the heart, each beat seeming to increase the pain in the head. Brought on by exposure to the sun.

*Ignatia.*.—Hysterical headache; clavus hystericus; from notional excitement or over-pressure at school. Highly nervous temperaments.

*Iris.*.—The attack begins with dimness of vision and terminates with the vomiting of a yellowish, bitter, sour-smelling fluid. Usually right-sided.

*Pulsatilla.*.—Left-sided attacks, with anorexia, belching and vomiting. Anæmia in mild, yielding subjects.

*Sanguinaria.*.—Pain beginning in occiput and spreading over the top of the head, settling over the right eye. Great sensitiveness to light; flushes of heat and alternate chilliness. The attack ends in vomiting.

*Spigelia.*.—Neuralgic pains over the left eye Chlorosis.

## CHAPTER XVII.

### DISEASES OF THE EAR, NOSE AND THROAT.

#### OTITIS.

Inflammation of the middle ear is of common occurrence during infancy and childhood, although it is a condition that is frequently not suspected unless an ear discharge appears. Being often followed by most serious sequelæ, which may either result in the death of the patient or leave him permanently deaf for the remainder of his life, it is of the greatest importance that its advent should be anticipated; that it be recognized early and the proper treatment instituted during the course of the attack. Every case of otitis, however, does not present so serious a prognosis. As will be seen from the description of the course of the different varieties, there is a mild, catarrhal form complicating rhino-pharyngitis or apparently occurring primarily and also a serious suppurative variety occurring as a complication of one of the infectious fevers, notably scarlatina.

The external auditory canal is directed more forward in the infant than in the adult, for which reason it is at times necessary to draw the lobe of the ear downward and forward in order to insert the speculum instead of drawing the aurical upward and backward, as in adults. Often the speculum is best inserted when traction is made directly backward on the aurical, and I cannot say that, as a rule, I find the direction of the canal forward, as mentioned in most text-books.

The Eustachian tube is wider, shorter and more horizontally placed than in the adult, and this anatomical feature, in conjunction with the prone position so constantly assumed by the child, offers the explanation why extension of an infection of the nose and throat travels so readily through the tym-

panum. The tympanic orifice is larger than the pharyngeal. Inflation of the middle ear is more easily accomplished than in adults.

The membrana tympani, or drum head, is almost horizontally placed, at first gradually assuming the perpendicular as the ear develops. It is thicker than in the adult and does not rupture so readily spontaneously.

The tympanic cavity is bounded superiorly by a thin plate of bone upon which the middle lobe of the brain rests. In the infant a suture, the petroso-squamosal, is found, allowing a vascular communication between the middle ear and the dura mater. For this reason meningeal irritation is so commonly observed in conjunction with otitis media. The close proximity of the inferior wall to the jugular fossa accounts for the liability of phlebitis and thrombosis of the jugular vein to occur as complications.

The upper portion of the tympanic cavity containing the malleus and part of the incus is known as the attic. It communicates with the mastoid antrum, and for this reason an accumulation of pus in the tympanum reaching to or confined to this point is usually followed by infection of the mastoid process. On account of the undeveloped state of the mastoid, however, involvement of the petrous bone and of the brain is more common than mastoiditis.

The mucous membrane lining the tympanum is quite thin and vascular, presenting a reddish and swollen appearance in young infants.

The mastoid process is but a small, undeveloped tuberosity at birth and contains, as a rule, only one cell, the antrum. It gradually develops by extending downwards and at the age of five years reaches the adult type. The upper wall of the antrum is in close proximity to the dura mater, being separated therefrom by only a thin lamina of bone.

The facial nerve passes along the upper portion of the tympanic cavity and downward through the mastoid cells. For this reason it frequently becomes affected in middle ear and mastoid disease.



**Earache** is the most prominent symptom of otitis, but it is possible for an inflammation of the middle ear to exist without any definite pain. This sometimes occurs in marantic infants, in whom an ear discharge may be the first sign of the trouble. Again, the pain may be vague and not definitely localized or be masked by cerebral irritation, but in these cases pressure at the tragus will usually elicit tenderness.

Tenderness and redness (inflammatory blush) over the mastoid indicates involvement of the mastoid cells and is an unfavorable symptom.

**Discharge.**—In the acute forms of otitis media that lead to perforation of the membrana tympani the discharge at first is serous as a rule, becoming muco-purulent later on. In the severe form, namely, that complicating *scarlet fever*, it is usually purulent from the beginning; the ordinary catarrhal variety, however, may assume a purulent character if its course becomes protracted.

**Tuberculosis.**—In the tuberculous variety of otitis the mucous membrane of the tympanic cavity is pale and the discharge is watery or a thin pus, in which the tubercle bacillus may be demonstrated.

**Influenza.**—A large number of cases of otitis are due to infection with the influenza bacillus. In these cases the discharge at first is sero-sanguinolent, later becoming sticky. There is always more or less blood, on account of the great congestion of the mucous membrane of the tympanum and of the drum head.

#### ACUTE CATARRHAL AND ACUTE PURULENT OTITIS MEDIA

The two varieties will be considered under the same heading, as it is impossible to draw a sharp line of distinction between them. Frequently what in the beginning seems to be a catarrhal otitis eventually becomes a purulent one and again, the early symptoms of both varieties are almost identical. This much, however, may be said, the catarrhal variety is by far the commoner in infants, while in older children the

purulent variety predominates. The explanation of this lies in the fact that catarrhal otitis usually develops secondarily to an acute naso-pharyngitis, while the purulent variety develops in the course of one of the infectious diseases, notably, scarlet fever and measles, and less frequently in typhoid fever, pneumonia and diphtheria. Influenza is a common cause of the more severe catarrhal cases. The micro-organisms most commonly found in the discharge are the pneumococcus and the streptococcus; the latter is responsible for the damage done to the middle ear and adjacent structures in scarlatinal otitis and the other grave symptoms of suppurative otitis. As a predisposing cause adenoid vegetations stand most prominently.

**Symptomatology.**—In infants otitis is usually preceded by a naso-pharyngitis; as the ear becomes involved there is an increase of fever and earache sets in. Although the child frequently gives evidence of the seat of the pain by putting the hand to the side of the head and by crying when the affected ear is touched, still there are a great many cases in which earache is not suspected until the membrana tympani has ruptured and a discharge makes its appearance. This is especially the case when otitis complicates an acute illness, such as pneumonia for example. In these cases there will be a rise of temperature that cannot be accounted for and the child will cry incessantly for no known reason. In the course of a day or two the appearance of the ear discharge clears up the mystery. Sudden exacerbation of fever in any acute illness not accounted for by other complications should always lead to an examination of the ears.

The crying of earache is characteristic. When we are confronted with an infant that is crying continuously in spite of every effort that may be made to make it comfortable, and, if other causes can be excluded, there is every reason to suspect earache.

In older children the disease is ushered in with excruciating pain and high elevation of temperature. Pain begins in

the ear, but radiates practically over the entire side of the head. As a rule, it is promptly relieved when perforation takes place.

Often the symptoms closely resemble meningitis, the disease is ushered in by convulsions and vomiting, and marked cerebral irritation is present on account of the close connection between the middle ear and the dura mater. These symptoms, however, disappear as soon as the middle ear is evacuated.

Many subjective symptoms are complained of, but the most important ones of the disease are those above referred to.

Early in the disease the drum head in the region of Shrapnell's membrane is congested. There is also hyperæmia extending along the posterior border of the handle of the malleus; the drum-head loses its lustre and assumes a deep pink color varying with the intensity of the inflammation. The external auditory canal also becomes deeply congested.

At first the drum head is somewhat depressed, but as the exudate fills the tympanic cavity it bulges, especially in its posterior half. When perforation occurs it most frequently takes place in the lower anterior or posterior quadrant of the membrane. Spontaneous perforation is less apt to drain the tympanum as thoroughly as an artificial puncture, nor does it heal as well.

When the pain continues after perforation, we should suspect involvement of the periosteal layer, or of the mastoid cells.

The complications of otitis media are mastoiditis; facial paralysis; meningitis; cerebral abscess; septicæmia; thrombosis of the lateral or other sinuses; caries of the sinuses; facial erysipelas and eczema aurium. As has been stated above, affections of the petrous bone and of the brain are commoner complications than mastoid disease in children. When the hearing is lost as a consequence of otitis, in the very young, deaf-mutism supervenes.

**Prognosis.**—There are two factors influencing the prognosis of otitis in children. In the first place the constitution

and the state of health at the time the disease is contracted are important factors. Secondly, the nature of the causative infection is important; those complicating a simple rhinopharyngitis or influenza are not as grave as those complicating scarlet fever (streptococcic) or those due to the pneumococcus or to the diphtheria bacillus. Again, the development of a complication augurs an unfavorable prognosis, and practically makes it a surgical condition. The prognosis is always more favorable when early incision of the drum-head has been made and free drainage established.

**Diagnosis.**—Earache should always be suspected when an infant cries continuously or when the fever suddenly rises during the course of an acute illness without assignable cause. The throat must be examined for evidences of pharyngitis and when enlarged tonsils and adenoids are found the possibility of earache should never be lost sight of. Inspection of the ear drum will give positive evidence of the disease.

**Treatment.**—Absolute rest in bed should be enforced and much relief of suffering may be obtained by instilling hot water into the external auditory meatus, or, better, by the instillation of a 10 per cent. solution of Carbolic acid crystals in glycerin in the hope of aborting the attack by osmotic action through the membrana tympani. Fill the external canal every two to three hours with a 10 per cent. solution of crystals of acid Carbol. in glycerin. This is not only useful in relieving the pain, but will at times abort the attack by osmotic action through the membrana tympani, and in any case it will render the canal aseptic in anticipation of perforation, natural or artificial (C. M. Thomas).

The most important remedies are *Aconite*, *Belladonna* and *Pulsatilla*. Even the old-school places great confidence in these remedies. Thus, Bacon (*Manual of Otology*) says: "Aconite in drop doses is a most valuable remedy when there is fever and especially in cases due to cold. Tincture of *Pulsatilla*, likewise given in drop doses, is indicated also in cases in which there is a profuse discharge from the nares or

naso-pharynx, and may be administered alternately with *Aconite*." The nose and throat should also receive attention.

When these measures fail to give relief, and if the fever and concomitant symptoms persist, the next indication for treatment is to freely incise the drum-head. Thomas (*Hahnemannian Monthly*, Oct., 1901) lays down the following rule: the acuter the attack and the more severe the suffering and prostration, the earlier should this operation be done.

A successful paracentesis is a free incision of the membrane and not merely a puncture. The technique is as follows: the patient having been anæsthetized and the external auditory canal thoroughly cleansed with a hot 1-5000 bichloride of mercury solution, the drum-head is inspected with the aid of a speculum and head-mirror in order to determine the site

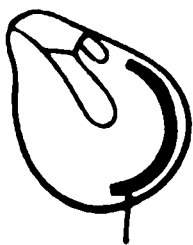


FIG. 51.—LINE OF INCISION  
THROUGH DRUM-HEAD  
(AFTER BACON).

of bulging if this be demonstrable. The incision is made with a narrow bistoury or tenotome. Ordinarily the line of incision extends from just behind the stapes to the lower border of the drum-head, closely hugging the bony structure of the canal (Fig. 51). In grave cases, with

bulging of the drum-head in its posterior and upper quadrant, together with indications of mastoiditis, the incision should be carried well up the posterior fold and into the attic. At the same time the knife should be brought out along the upper posterior wall of the external auditory canal to relieve all tension. The canal is then lightly packed with sterile gauze and after the acute symptoms have subsided irrigation with 1-5000 bichloride solution may be practiced several times daily. If the discharge persists Thomas recommends the instillation of a saturated solution of *Boric acid* in alcohol, followed by: *Zinc. sulph.*, *Acid carbol.*, āā, grs. 5; aqua distil.; alcohol, āā oz.  $\frac{1}{2}$ ; eight to ten drops instilled after cleansing, three times daily. Inflation, cautiously employed, when the perforation is large, helps to remove the secretion from the tympanum.

*Remedies.*—In the acute stage, *Aconite* and *Pulsatilla* are most commonly indicated (see above). *Belladonna* is the remedy when cerebral symptoms are prominent. *Capsicum* comes highly recommended for the early stages of mastoid involvement.

During the period of discharge, *Pulsatilla* and *Calcarea iodid.* are most useful. *Hydrastis* is particularly indicated in influenzal cases, where the discharge is sticky and tenacious. When the discharge excoriates we should think of *Mercurius* and in involvement of the bone *Silica* is the most useful remedy.

As the discharge decreases the instillations should be made less frequently and finally daily dusting of the canal with *Boric acid* should be substituted. After cessation of the discharge and closure of the perforation the restoration of hearing will be greatly hastened by cautious inflation with the Politzer bag or catheter, every one to two days (THOMAS).

#### ACUTE TONSILLITIS.

Acute inflammation of the tonsil may be either superficial, or catarrhal; folliculous, or cyptic; and parenchymatous. Anatomically the tonsils consist of an aggregation of lymphoid tissue embedded in connective tissue and covered by a mucous membrane from whose surface numerous mucous glands dip into its parenchyma. These glands form the so-called crypts, or follicles, and they play an important role in the diseases of the tonsil.

Clinically the tonsil is most important as the port of entrance of the infective agent of many of the infectious diseases. Diphtheria and scarlet fever notably attack the tonsils, and rheumatic fever is now looked upon as frequently beginning as a tonsillar infection. Indeed, a special variety of tonsillitis designated "*rheumatic tonsillitis*" is described by some writers, but such a condition should be, strictly speaking, looked upon as an attack of rheumatic infection in which tonsillar symptoms predominate, for in these

cases, especially in children, a careful examination of the case often reveals the presence of endocarditis and tenderness in the joints.

**Acute Superficial Tonsillitis.**—As the name implies, acute superficial tonsillitis involves only the mucous membrane covering the tonsil, but as a rule deeper structures are more or less involved. The process may also spread to contiguous structures, and it either undergoes prompt resolution or in the case of secondary infection is followed by superficial necrosis of the epithelium, or suppuration of the connective tissue takes place, resulting in peritonsillar abscess.

It is a common accompaniment of many of the infectious diseases, notably measles and scarlet fever. In primary cases the usual etiological factor is “taking cold,” and by many it is believed that the “rheumatic diathesis” offers especial predisposition to these attacks.

**Symptomatology.**—In primary cases there is malaise and slight chilliness, together with dryness of the throat and more or less pain on swallowing. The tonsils appear bright red, swollen, and their surface presents a somewhat oedematous appearance. It is seldom that the process ends here, however, the crypts usually becoming occluded and filled with fibrin, leucocytes and epithelial debris, which constitutes acute folliculous tonsillitis.

Associated symptoms are fever; headache and malaise; stiffness of the neck, even torticollis and earache.

#### ACUTE FOLLICULOUS TONSILLITIS.

Acute folliculous, or *cryptic* tonsillitis, is an acute infection of the tonsils. The germs usually found are the streptococcus, staphylococcus, and pneumococcus. In many cases there is associated superficial necrosis of the mucous membrane covering the tonsils, together with an exudation of fibrin and the formation of irregular patches of pseudo-membrane. This condition is a frequent complication of scarlet fever, although it may occur independently. It is known as “pseudo-diphtheria.”



**Symptomatology.**—The attack begins with malaise and creepy sensations, usually along the spine, followed by fever and aching throughout the body. There is dryness of the throat and some pain on swallowing, but frequently the child does not refer to its throat until the tonsils are greatly swollen, and one is often led to look upon the condition as influenza or beginning typhoid fever unless the routine examination of the throat is practiced.

Fever persists for about three days, together with an increment in the severity of the symptoms, ranging between 100° F. to 105° F. By this time the inflammation of the tonsils has reached its climax and they present a characteristic appearance. They are deeply congested, uniformly swollen and their surface is studded with yellowish-white, punctate spots appearing at the mouths of the crypts. When the exudation is abundant it spreads over the surface of the tonsils and may give rise to the appearance of a membrane. This is, however, readily wiped off. Again, necrosis of the epithelium around the mouths of the crypts frequently takes place, the spots assuming an irregular outline, like a diphtheritic membrane, and these spots may coalesce; but the deposit is only superficial and is readily wiped off, distinguishing it from diphtheria.

The lymphatic glands of the neck may become enlarged and tender, but never to the extent found in diphtheria.

Associated symptoms are painful deglutition—in fact pain at the height of the disease is one of its most characteristic symptoms; lancinating pains extending into the ears; headache and prostration.

The tongue is coated and slimy; the breath is offensive, but nothing like in diphtheria, and there is anorexia and constipation.

The fever subsides on about the third day; the tonsillar swelling abates at the same time, and convalescence is established in the course of a few days.

**Diagnosis.**—The most important condition from which

folliculous tonsillitis is to be distinguished is diphtheria. In a typical case this is comparatively easy, but in the class of cases described as pseudo-diphtheria many difficulties are encountered. High fever, occurring suddenly in older children, is perhaps most frequently due to tonsillitis.

The characteristic points to be remembered in the diagnosis of folliculous tonsillitis are : The punctate spots of soft, unorganized exudation confined to the tonsillar crypts; the uniform inflammation and swelling of the tonsils; the high fever and pain and the absence of profound toxæmia; and, lastly, the absence of marked enlargement of the lymphatics of the neck. In all doubtful cases, however, a bacteriological examination of the exudate should be made, for in rare instances the diphtheria bacillus sets up a tonsillitis identical in appearance with the ordinary folliculous variety.

**Treatment.**—If there be fever the child should be put to bed and isolation of the patient enforced. When there is much pain and swelling of the tonsils an ice collar will give decided relief. The throat may be sprayed several times daily with a mild antiseptic, such as Asepticon (Boericke & Tafel) diluted with warm water, and when there is considerable exudate and offensive breath the *Permanganate of Potash*, 1 to 1,000 solution, is preferable.

The most important **remedies** are : *Belladonna*, *Mercurius iod. rubr.*, *Apis* and *Ignatia*.

*Belladonna* is indicated in the early stage when there is dryness and redness of the throat with pain on swallowing; throbbing headache; photophobia; high fever and flushed face. It is more frequently indicated in tonsillitis in children than in adults.

*Apis* is indicated when œdematous swelling of the mucous membrane is the leading feature in the case. There are sharp, sticking pains on swallowing.

*Ignatia* is a valuable remedy in folliculous tonsillitis when there are sharp, lancinating pains extending into the ears.

*Mercurius iod. rubr.* is the most useful remedy in the fully developed stage, especially when exudation is abundant.

## ULCERO-MEMBRANOUS TONSILLITIS.

This is a condition presenting a marked outward resemblance to diphtheria, but on close study it will be seen that the resemblance is merely superficial. In ulcero-membranous tonsillitis the tonsil becomes covered with a dirty-yellowish exudate; this is often confined to a single tonsil. When the exudate is wiped away, especially when done roughly, a bleeding surface may remain. The lymphatics at the angle of the jaw on the affected side are swollen. Thus far there is a strong resemblance to diphtheria, even to offensive breath, but constitutional symptoms are slight or wanting and a bacteriological examination reveals instead of the Klebs-Loeffler bacillus the fusiform bacillus discovered by Vincent and supposed to be the etiological factor. Pseudo-diphtheria is also to be differentiated (see Diphtheria).

Ulcero-membranous tonsillitis is at times associated with ulcerative stomatitis and is looked upon as being an analogous condition.

The *treatment* is the same as for other forms of tonsillitis. Locally, *Hydrogen dioxid*, preferably as a spray, is the most useful disinfectant. The red *Iodide of Mercury* is well indicated as an internal remedy. For fuller symptomatology see "Tonsillitis."

*Merc. iod. rubr.*—This is the most useful remedy in cases resembling diphtheria where there is superficial ulceration of the tonsils; fibrinous exudation and enlargement of the cervical lymphatics.

## ACUTE PARENCHYMATOUS TONSILLITIS; PERITONSILLAR ABSCESS.

Acute parenchymatous tonsillitis, commonly called "quinsy," results from an infection of the tonsil from without, either following superficial ulceration or associated with a membranous or inflammatory process of the tonsil and surrounding structure; it may also be secondary to some

other form of tonsillitis and to the infectious diseases ; and it may be associated with systemic septic processes (KYLE).

Suppuration as a rule sets in, taking place in the peritonsillar connective tissue and terminating in the formation of an abscess which may rupture into the pharynx either anteriorly or posteriorly, following the line of least resistance. It is a disease common in later childhood and in adolescents.

**Symptomatology.**—The onset is similar to that of other forms of tonsillitis, with the exception that the inflammation is one-sided and attended with more pain and swelling. The pain at first is lancinating ; later it becomes throbbing in character. There is a constant desire to swallow, which adds greatly to the discomfort of the patient. Fever and malaise are usually not so marked as in folliculous tonsillitis.

On inspection, the throat presents a swollen, œdematous appearance and a tumefaction arising from the tonsillar region is seen projecting toward the median line. The tonsils and pharynx are covered with a grayish, viscid mucus which gives the appearance of a thin pseudo-membrane being present, but by spraying the throat it can be completely removed. The tonsil itself is not the seat of the chief swelling, but it is simply carried into the median line by the surrounding tumefied structures. The opposite side may become affected later on, but the disease is rarely bilateral. Inspection is difficult on account of the stiffness of the jaw that is associated. Fluctuation may be elicited, but it is not always easy to determine on account of the boggy, œdematous condition of the tissues.

The duration is from a few days to a week or longer. Resolution may set in, or spontaneous evacuation take place after four or five days with prompt relief of the symptoms.

**Treatment.**—If suppuration cannot be aborted by the use of the ice-bag and the indicated remedy, the abscess should be evacuated as soon as pus is suspected and an antiseptic gargle freely used. The incision is made with a sharp pointed bistoury whose cutting edge has been wrapped in

cotton, exposing only the point for a distance of about a quarter of an inch. The point is inserted to its full length into the substance of the half arch just above the tonsil and a quarter of an inch from its free border, and the tissue cut through and across, toward the median line. Peritonsillar abscess can often be most satisfactorily evacuated by passing a bent probe outward and upward posteriorly to the anterior half and into the supra tonsillar fossa (THOMAS). The patient should then gargle with a warm 2 per cent. *Boric acid* solution, or preferably diluted *Hydrogen dioxid* so long as pus is present.

**Remedies.**—*Belladonna* in the early stage; later, as soon as pus begins to form, *Mercurius vivus*; and *Hepar sulph.* to hasten resolution, are the remedies that will be needed in the majority of cases.

*Apis* may become indicated from a predominance of œdema. In fact, œdema of the glottis may supervene, and for this condition *Apis* is looked upon as invaluable.

*Capsicum*.—Serous infiltration of the faucial tissues; boggy not œdematous, in appearance; left side worse; pain, burning, stinging. When tongue is heavily coated white, uvula œdematous, especially with a dusky infiltration of the left pillars and some swelling of the lymphatic glands, *Caps.*, in the 3x or 6x, will usually relieve inside of twenty-four hours (IVINS).

*Guaiacum*.—Recurring attacks due to rheumatic diathesis.

*Phytolacca*.—Chills and fever alternate; prostration; pain running to ears on deglutition; affected parts dark-purple, almost blue; rheumatic subjects; uvula enlarged and œdematous.

*Silicea*.—Protracted cases. Suppuration continues after evacuation of pus has taken place (*Calc. sulph.*).

#### HYPERTROPHY OF THE TONSILS.

There are two varieties of hypertrophy of the tonsils; in the one the increase in structure is mainly glandular, while

in the other it is interstitial. The first variety is known as the soft, glandular type; the other as the hard, fibroid, or lobulated tonsil. An enlarged tonsil is not necessarily an hypertrophied one, as enlargement may result from vascular engorgement and does not necessarily indicate cell proliferation. Again, in children the tonsils are normally large, and because they extend beyond the pillars of the fauces, it does not necessarily follow that they are hypertrophied (KYLE).

The *cause* of the various enlargements is both constitutional and acquired. The so-called strumous diathesis, or what is understood by the more modern term lymphatism, is the underlying constitution looked upon as responsible for the abnormal tendency to hyperplasia of these lymphoid structures. Recurring attacks of acute tonsillitis, and diphtheria and scarlet fever may be looked upon as the chief exciting causes. The condition belongs practically to the period of childhood.

**Symptomatology.**—Subjective symptoms depend largely upon the size of the tonsils. They may be so large as to cause considerable interference with normal respiration by filling up the pharyngeal space, and under these circumstances the voice is also affected, acquiring a nasal twang. Many of the symptoms resulting from adenoid vegetations are also caused by enlarged tonsils.

In the soft variety the tonsil is uniformly enlarged, while in the fibrous variety it is lobulated; the crypts are abnormally large, and its consistency is hard and unyielding.

The irregular, nodular surface of the enlarged tonsil; its open crypts and eroded surface; and its perverted function, render it a source of danger as an avenue for infection, beside its other evil effects upon the child's health.

**Treatment.**—Unless the tonsils are sufficiently enlarged to interfere with the child's health, or to affect the voice, they will require no further treatment than mild local measures and a remedy prescribed upon a constitutional basis. When local symptoms are marked the remedy should be chosen on such indications. It is the simple, hypertrophic variety of

enlarged tonsil without connective tissue proliferation that so promptly improves under appropriate treatment and undergoes physiological atrophy in later life. The fibroid variety, however, is rarely improved by treatment of any kind, and if it be large enough to cause symptoms it should be excised.

Excision can be accomplished in older children under local anæsthesia (a 4 per cent. solution of *Cocaine*), but in the young this should not be attempted. The same preparations as for the operation for adenoids are made, and under good illumination the tonsils are cut off with the tonsillotome close to the pillars of the palate. When adhesions between the tonsils and palatine folds exist they should be broken up with a blunt instrument, such as the Allis dry dissector, before removing the tonsil.

Bleeding is rarely alarming, and it can be controlled by pressure with a mop dipped in a saturated solution of *Tannic acid*. Under anomalous circumstances a hæmorrhage may ensue; this will require long continued pressure (with the finger or an especially constructed tonsillar hæmostat), or it may become necessary to place a ligature around the stump of the tonsil. The after treatment is the same as for adenoids.

**Remedies.**—Of the many remedies recommended for enlarged tonsils there are only a few that have given me positive results. They are *Calc. phos.* and *Ignatia. Baryta carb.*

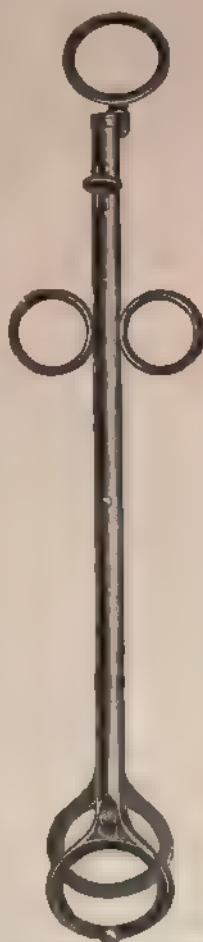


FIG. 52 TONSILLOTOME.



and *jod.* are usually prescribed for the class of enlargement that is beyond the pale of medicinal action ; consequently the results I have seen from them are not gratifying. *Ignatia* is of undoubted value in the early stages of simple glandular hypertrophy, and it especially suits those cases in which there is a constant recurrence of acute tonsillitis. *Calc. phos.* is the constitutional remedy best suited to the condition that predisposes to the overgrowth of the lymphoid structures and its efficacy in tonsillar hypertrophy is not to be questioned. If taken in time most cases will no doubt escape operation, but one should not be too sanguine of results in those of long standing.

#### RETRO-PHARYNGEAL ABSCESS.

The commonest variety of retro-pharyngeal abscess is the result of an acute infection of the lymphatic glands and vessels of the pharyngeal space ; in other words, an acute lymphangitis and adenitis that has broken down in suppuration. A *septic* variety, occurring as a complication of scarlet fever and measles, is sometimes encountered, but it is much rarer than the idiopathic form. *Chronic retro-pharyngeal abscess* is due to cervical Pott's disease. This occurs in childhood, while the above condition occurs almost exclusively during infancy.

As the lymph-nodes of the retro-pharyngeal space are intimately connected with the lymphatics of the tonsils and uvula, any acute inflammatory condition of these structures is likely to result in involvement of the pharyngeal lymphatics. This is especially the case during the period of infancy, when these glands are in a state of high physiological activity. Later in childhood, however, they atrophy, for which reason retro-pharyngeal suppuration is rare after the third year.

The tumefaction may be situated in the median line, but more frequently it is more to the side and may even appear to arise from behind one of the half-arches. The glands at

the angle of the jaw may also be implicated, in which case the swelling is found at or beneath the angle of the jaw and in front of the sternocleido-mastoid muscle. In such cases a spontaneous evacuation of the abscess externally may take place, although the majority break into the pharynx.

Septic retro-pharyngeal abscess complicating scarlet fever and measles shows a tendency to burrow into the mediastinum or ulcerate into the carotid arteries and other important structures.

**Symptomatology.**—The onset is insidious and usually it is not suspected until marked symptoms have developed, as there is present always a primary inflammatory condition of the nose or throat upon which it depends. In the course of five or six days, by which time the primary condition should have entirely subsided, there is still a trace of febrile movement and inspection of the throat reveals a swollen and œdematous state of the pharyngeal mucous membrane. Two or three days after this, evidence of suppuration becomes apparent and the swelling has attained such size as to call forth the symptoms characteristic of the disease. There will be difficulty of breathing, especially on inspiration; crowing respiration, due to inco-ordination of the vocal cords; retraction of the head in order to give the larynx as much free space as possible and distinctly nasal cry. The child breathes with the mouth open and holds the head so rigid that cervical Pott's disease or torticollis may be erroneously thought to exist. Inspection of the throat will, however, immediately clear away any doubt as to the true nature of the case. The abscess is readily made out by carefully introducing the index finger into the pharynx. This must always be done with caution to avoid rupturing the abscess or throwing the infant into collapse by rude manipulation of the fauces.

If allowed to rupture spontaneously the pus may be aspirated into the lungs, causing instant death or setting up a fatal broncho-pneumonia; it may also find its way into the Eustachean tubes and set up an acute otitis. In many in-

stances, however, the pus is swallowed or evacuated through the mouth without causing any trouble. Nevertheless, prompt surgical interference offers the best prognosis and should be instituted in all cases as soon as they give indications for the evacuation of pus.

**Treatment.**—The abscess is easily incised when it points to the median line or not far therefrom. Cases in which the swelling is well to the side require great care, as there is danger of wounding the carotid artery. Those pointing externally must be opened with great care, as deep incision must be made in order to thoroughly drain the abscess. Tuberculous abscesses should be opened externally whenever possible.

The child is held firmly in the upright position and the throat illuminated by the head-mirror. A mouth-gag is unnecessary; all that is required to expose the abscess and keep the mouth open is a reliable tongue depressor. The incision is made toward the median line with a bistoury whose cutting edge has been protected by wrapping it with cotton up to within half an inch from the point. After making the incision it is often necessary to break up septa of connective tissue within the abscess cavity with the tip of the index finger.

The remedies indicated are *Belladonna* in the early stage and *Hepar sulph.* when pus begins to form.

#### ACUTE RHINITIS; PSEUDO-MEMBRANOUS RHINITIS.

Acute rhinitis is an acute inflammation of the mucous membrane of the nasal cavities occurring either as a primary condition or secondary to one of the infectious diseases, notably measles, influenza and diphtheria; the cause of the acute suppurative symptoms of rhinitis lies in infection by pyogenic germs which are usually found present in the nose in great number. They do not, however, become active until the vascular engorgement of the nasal mucosa resulting from exposure to cold or draughts offers a favorable soil for their propagation, and invites them to activity.

*Pseudo-membranous rhinitis* associated with faucial diphtheria is due to the *Klebs-Löffler bacillus* in its most virulent form, while those cases in which a diphtheritic membrane develops primarily in the nose, running a mild course, the bacillus is present in attenuated form. Such cases, however, may give rise to a severe faucial diphtheria, and for this reason every case of pseudo-membranous rhinitis should be isolated. This attenuated diphtheria bacillus is known as Von Hoffman's bacillus. According to Park (*Bacteriology in Medicine and Surgery*) only in a few cases have other bacteria been found to cause the croupous exudate; they were mainly the pyogenic cocci. Kyle (*Diseases of the Nose and Throat*) is of the opinion, however, that most cases of croupous rhinitis are simply the result of local irritation from micro-organisms, the streptococcus pyogenes being the most frequent one present in the croupous exudate. All of the cases that have come under my notice were diphtheritic. At times it seems due to some constitutional condition in which the individual cell resistance is below normal. It may also result from traumatism.

There is no doubt that a certain amount of contagiousness exists in acute rhinitis. A natural predisposition is found in many cases; this is particularly the case in anæmic children that have been reared like hot-house plants and in those of the so-called scrofulous diathesis.

**Symptomatology.**—Following upon exposure, or “catching cold” or in the course of an infectious disease a sense of fulness in the nostrils with dryness of the mucous membrane develops, succeeded by an acrid, watery discharge consisting of serum with a small amount of mucus. At this stage the mucous membrane appears red and swollen, and the entire nasal cavity may be occluded by the swollen turbinated bodies.

In primary cases a slight febrile reaction sets in and there is headache, and lassitude. Mild cases may be aborted at this stage and resolution occur without any further develop-

ments. In infants these attacks are spoken of as *snuffles*, and unless they are due to syphilis or are benign, profuse mucopurulent secretion makes its appearance, flowing freely from the nose and covering over the entire mucous membrane of the naso-pharynx. The process may extend to the frontal sinuses, the Eustachian tubes and middle-ear, and to the pharynx. If the infection has been of a virulent nature ulceration of the mucosa and suppuration of the middle-ear are liable to supervene.

**Pseudo-membranous rhinitis** is almost invariably diphtheritic in origin, as has been stated above. From the fact that constitutional symptoms are usually slight in primary diphtheritic, or fibrinous rhinitis, it frequently remains unsuspected until the membrane is accidentally discovered. The membrane may persist for weeks, coming away in large pieces. If during its course it be removed, it usually recurs. The nose is more or less obstructed, and a thin blood-streaked discharge runs from the anterior nares. Such a secretion should always arouse suspicion of diphtheria. On inspection, the membrane is seen as a firm, grayish exudate upon the interior of the nose. The disease is far more benign than faucial diphtheria with or without extension of the membrane to the nose, but it may assume a most unfavorable course by spreading to the pharynx, under which circumstance severe constitutional symptoms will arise.

**Treatment.**—In the early stages the obstruction may be much relieved by spraying or douching the nose with a warm mild alkaline antiseptic solution, such as Dobell's solution, or a normal saline solution, followed by spraying with a bland oil containing camphor or menthol in the proportion of one grain to the ounce. Later as the discharge becomes profuse, frequent cleansing of the nasal passages is imperative. In infants or young children who struggle against the use of the atomizer, a small glass syringe may be employed, injecting into one nostril and allowing the fluid to flow out of the other, the child lying on its side during the operation.

In *pseudo-membranous rhinitis* a 1 to 1000 solution of permanganate of potash should be used freely in the form of an irrigation, allowing about a pint to run through the nares at intervals of a few hours (see *Nasal Syringing*, Chapter I).

In the early stages *Aconite* and *Gelsemium* are the most important remedies. In the snuffles of infants *Dulcamara* has given good results, and when associated with great embarrassment of respiration, causing the child to start just as it is falling asleep on account of the extreme nasal stoppage, even in the presence of free secretion, *Ammonium carb.* is a most valuable remedy. Hughes (*Manual of Therapeutics*) considers *Camphor* a specific in the early stage, promptly aborting most cases and especially relieving the chilly feeling.

*Aconite*.—Sneezing; fever with restlessness and full pulse; burning of the eyes.

*Gelsemium* differs from *Aconite* in the absence of the restlessness and high arterial tension and in the predominance of malaise; chilliness, especially creeps up and down the spine but not a well defined chill; headache with drowsiness and heaviness of the eyelids; aching in the muscles. *Gelsemium* colds are such as are contracted during warm moist weather or occurring in debilitated subjects; the *Aconite* cold typically occurs in active, plethoric individuals after exposure to cold winds.

*Nux vomica* is indicated in the early stages of many cases; there is dryness and obstruction of the nose; fulness at the root of the nose and frontal headache; cold hands and feet with a hot head; anorexia and constipation; irritability of temper and feverishness. Subjects who are overly sensitive to draughts. In this respect

*Arsenicum* is similar. "Persons who are rarely without a cold" (IVINS). Sneezing; profuse, watery, excoriating discharge; tendency of cold to travel down upon chest.

*Belladonna* has always been a most satisfactory remedy in my hands for the vascular engorgement of the turbinated bodies. The mucous membrane appears dry and bright red and the nose is much obstructed.

*Cepa*.—Profuse, acrid watery discharge with lachrymation.

*Euphrasia* has a profuse nasal discharge which is bland but an excoriating lachrymal discharge, the opposite condition of *Cepa*.

*Ferrum phos.* is a valuable remedy in the early stages of coryza, being similar to *Aconite*, but without the feverish restlessness of that remedy. Given over an extended period of time it will do much to eradicate the cold-catching tendency.

*Sanguinaria Canadensis* or *Sanguinaria nitr.*, 3x trit., is useful when there is a sensation of great dryness and burning in the nose and pharynx, with headache and loss of smell and taste.

In the second stage, when the discharge becomes profuse and muco-purulent in character, no remedy is more useful in the majority of cases than *Pulsatilla*. When there is much soreness of the nose and evidence of ulceration *Mercurius* is the better indicated remedy. *Hydrastis* should be thought of, but it seems more useful in chronic cases.

*Pseudo-membranous rhinitis* requires the remedies useful where croupous exudation is found. *Hepar*, 3x trit., and *Kali bichromicum*, 2x trit., will most frequently be of service. Local treatment as directed above is not to be neglected. A culture should be made and if it verifies the presence of the Klebs-Loeffler bacillus, antitoxin should be used.

#### SIMPLE CHRONIC RHINITIS AND PURULENT RHINITIS.

Chronic rhinitis without pronounced hypertrophic or atrophic changes in the nasal mucous membrane is a common affection of childhood. Abundant muco-purulent secretion is usually associated with the catarrhal process and makes the disease a particularly unpleasant one.

In the etiology recurrent attacks play an important role. The period of childhood itself invites catarrhal inflammation with epithelial cell proliferation, the rapid desquamation of which constitutes the main pathological process in purulent rhinitis. It is not confined to those of the syphilitic or



scrofulous diathesis, apparently healthy constitutions falling victims of the disease as well as others. As a predisposing cause, adenoids undoubtedly play the most important role. Unhygienic surroundings, and want of attention during acute attacks or failure to guard against the recurrence of such attacks are the chief exciting causes. No specific micro-organism is present, but there is no doubt that an infection of a mixed character causes the purulent inflammation. Irritation by foreign bodies or other sources of irritation may induce similar pathological changes.

**Symptomatology.**—The chief symptom is a profuse mucopurulent discharge. Nasal obstruction is not pronounced. The nose may become reddened about the orifices and excoriated and crusts form in the anterior nares, usually at night, in this way inducing mouth breathing during sleep. Susceptibility to acute attacks seems lessened on account of reduced sensibility of the mucosa from loss of epithelial cilia (IVINS).

Atrophic changes will occur in the course of years if the progress be not arrested. It may also pass into the hypertrophic variety if rhinorrhœa has not been a prominent feature of the case. In scrofulous children infection of the cervical lymphatics is a frequent complication. In the majority of cases the prognosis is good, especially under proper treatment. Ozæna is the most unfortunate outcome that may be anticipated.

#### HYPERTROPHIC RHINITIS ; ATROPHIC RHINITIS.

Hypertrophic rhinitis is a chronic catarrhal inflammation of the nasal mucosa and sub-mucosa, characterized by hypertrophy of the turbinated bodies with resulting nasal obstruction. It is not as frequently encountered in children as in adults, nor is it as common a disease as atrophic rhinitis. The pathological changes are such as require a long time for their development, being a hyperplasia of the cellular elements and overgrowth of the connective tissue and blood-vessels that form the turbinated bodies.

A variety of hypertrophic rhinitis in which there is simply engorgement and dilatation of the blood-vessels is not uncommon. In this class of cases a complete temporary retraction of the mucous membrane may be induced by the local application of cocaine, or it may occur spontaneously or as the result of appropriate treatment.

*Atrophic rhinitis*, or *Ozæna*, is characterized by atrophy of the mucous membrane, of the cavernous structures, and the underlying bone. There is also atrophy of the mucous glands with consequent impaired function and the formation of offensive crusts. The crusts represent inspissated muco-purulent secretion which accumulates in the nasal chambers and undergoes decomposition. They are the cause of the fetor emanating from these patients.

**Etiology.**—Adenoid vegetations play an important role in the etiology of *hypertrophic rhinitis*, by interfering with the drainage of the nasal chambers, thus inviting the accumulation of irritating material which keeps up a constant congestion of the mucous membrane. Again, the constitutional peculiarity which invites adenoids and hypertrophy of the tonsils predisposes to chronic catarrh and hypertrophy of the intra-nasal structures. Clinically there is an intimate association of these conditions. Another cause will be found in recurrent acute attacks which may lead up to permanent structural changes.

*Atrophic rhinitis* may develop as an independent affection or as a sequel to hypertrophic rhinitis. Casselberry dissents from the latter view, believing the transition of an hypertrophic rhinitis an exceedingly rare, and in all events slow process; and he looks upon atrophic rhinitis, particularly in children, as a distinct affection. A pronounced hereditary predisposition, moreover, has often been observed. Bosworth believes suppurative rhinitis of children to be the cause of atrophic rhinitis, the suppurative process destroying the mucosa layer by layer in the course of time, until eventually the deepest structures become involved.

**Symptomatology.**—The chief symptom of *hypertrophic rhinitis* is nasal obstruction. This may be more or less complete and involve both sides simultaneously or alternately. Remissions occur, and frequently the nose will be clear under ordinary circumstances, only clogging up when irritated by the inhalation of dust; walking in the wind; entering a warm room, etc. This peculiar behavior readily explains itself when we remember that the obstruction depends upon the degree of vascular engorgement present at the time.

As a result of the reflex irritation in the nose and the interference with respiration, a train of symptoms indicating a disturbance in the general health of the child arises. Nervous irritability; disturbed sleep and mouth breathing; intellectual torpor; hæmicrania; spasm of glottis; asthma and enuresis, all may have their origin in the nasal stenosis. It is hardly possible to differentiate between the disturbances induced by hypertrophic rhinitis and those induced by adenoid vegetations; the latter, however, are likely to induce even graver troubles than the former, and they are more frequently encountered as an independent condition.

On inspecting the anterior nares we will find the turbinated bodies swollen and of a deep red color, the inferior turbinated being most readily seen and darker in color than the middle or superior. If there be much engorgement it will be impossible to see more than the inferior body and at the most the anterior half of the middle body without making an application of *Cocaine* to shrink the mucous membrane. Polypi are likely to be confounded with an hypertrophied turbinated body, but they are paler in color, are movable, and occupy a position between the turbinated bodies.

*Atrophic rhinitis* is characterized by the formation of crusts and fetor. Obstruction of the nares only occurs if the crusts are allowed to accumulate in large masses. They may occur simply as scales, or form in large horny masses, completely occluding the nasal chamber. These masses eventually soften by decomposition or cause necrosis of the underlying mucous

membrane, coming away in large masses and leaving an ulcerated surface behind. The fetor may be so intense as to render the patient's proximity unbearable. In the beginning the child may be annoyed by the odor, but eventually the sense of smell becomes so obtunded that it is not aware of the fetor. There may be a sense of distressing fulness in the nose when crusts accumulate, and the habit of constantly picking the nose is soon acquired. Epistaxis is soon a frequent accompaniment. The general health is naturally affected; hearing becomes impaired, and the sense of smell may be entirely lost.

Inspection reveals a spacious nasal cavity lined with a thin, smooth mucous membrane, covered with crusts. Its surface is studded with superficial ulcers. *Hereditary syphilis* is to be differentiated from atrophic rhinitis; in the former there is not a uniform distribution of the atrophic process, and there is deep ulceration and cicatrization. Perforation of the septum with sinking in of the nose is pathognomonic of syphilis.

The *prognosis* is not unfavorable. Under persistent treatment most cases in children recover, some in the course of a few months, others not yielding to treatment in less than a year or two. Syphilitic cases, if seen early before destructive changes have set in, respond promptly to appropriate local measures in conjunction with anti-syphilitic remedies.

#### TREATMENT OF CHRONIC RHINITIS.

In undertaking the treatment of a case of hypertrophic rhinitis we must first of all determine whether it is an independent affection or due to adenoid vegetations. Should the latter prove the case we must proceed to remove the adenoids as directed under the article on Adenoid Vegetations. If the condition has not advanced beyond the stage of vascular engorgement a cure usually ensues upon the removal of the adenoids. When permanent hypertrophy of the turbinated bodies has set in there is but one sure and permanent method of treatment that fulfills all the requirements of a safe and

radical operation, namely, burning away the redundant tissue with the galvano-cautery. The inferior turbinated body is the obstructing body in the majority of cases, and by burning a linear eschar along its entire length, applying the platinum knife at the posterior border and drawing it forward slowly, burning down to the bone, sufficient retraction is obtained to overcome the stenosis. The operation can be performed entirely painlessly with the use of a 4 per cent. solution of *Cocaine*. In the course of a week or two the opposite side should be operated upon in the same manner. A mild antiseptic alkaline solution, such as Dobell's solution, or a solution of Seiler's antiseptic nasal tablets, should be used to cleanse the nose both before and after the operation. During the healing process it may be used either in a douche or in an atomizer, several times daily, to be followed by an oily spray, such as the Thuya Oil Spray made by Boericke & Tafel.

Should hæmorrhage occur after the cauterization or in the course of a day or two, when the scab comes away, it can readily be controlled by spraying with a 5 per cent. solution of the 1-1000 solution of *Adrenalin*, or by spraying with 10 per cent. solution of *Tannic acid*. Packing is seldom required.

Milder cases, not requiring surgical interference, should receive local applications of *Iodine* and *Glycerine* (5 per cent.), made by means of absorbent cotton on a probe, about twice weekly, followed by spraying with thuya oil. Besides, the nose should be cleansed daily with the alkaline antiseptic, preferably by douching with the Birmingham nasal douche or any other similarly constructed appliance. It is always safer to follow the cleansing process with the oil spray to prevent catching cold.

In treating *atrophic rhinitis* the most rigorous steps for maintaining absolute nasal cleanliness must be taken. The free use of the douche bag is here to be instituted, and a pint of Dobell's solution should be allowed to flow through the nares at a time. This should be done twice daily. For the method of giving the nasal douche see page 19.

If hard crusts have formed that cannot be dislodged by means of the douche, *Hydrogen dioxid*, diluted twice with warm water, should be slowly injected into the nares with a blunt syringe; this so loosens them that they can be readily blown out. After the nose has been cleared a few drops of refined carbon oil with iodine (one grain to the ounce) should be dropped into each nostril with a medicine dropper (KYLE).

When eroded surfaces remain after the removal of the crusts a stimulating powder, such as aristol, should be insufflated. Syphilitic ulcerations are best controlled by the local application of a ten per cent. solution of *Nitrate of Silver*.

**Remedies.**—When well marked constitutional indications are present such remedies as *Calc. phos.*, *Calc. carb.*, the *Iodides*, *Hepar* and *Silicea* will give better results than remedies selected purely on local indications. *Pulsatilla* and *Hydrastis* are especially useful in *simple, chronic and purulent rhinitis*.

In *atrophic rhinitis* the *Chloride of Gold*, *Kali bichromicum*, *Mercurius corr.* and *Silicea* are the most important remedies, *Aurum* heading the list.

*Syphilitic affections* require *Mercury*, preferably the yellow iodide when the ulceration is confined to the mucous membrane. When the bones become affected *Aurum metallicum* is indicated. Ulceration of the septum calls for *Kali bichromicum*. Gummatus infiltration of the soft structures will require the *Iodide of Potash* in material doses, five grains, three times daily, being the usual dose necessary to effect a cure.

*Alumina.*—Thick, greenish-yellow nasal discharge; anosmia; mind sluggish; snapping in the ears when swallowing.

*Arsenicum iod.*—Delicate, tuberculous constitution; acrid discharge with burning in nose. Chronic purulent rhinitis.

*Aurum.*—Offensive discharge; soreness of bones of nose. Ozæna and syphilis. The metal seems best indicated in syphilis, while in ozæna the chloride is preferable.

*Calc. carb.*—Glistening redness of nasal mucosa; extreme

sensitiveness of nose ; purulent discharge. Chronic purulent rhinitis in scrofulous individuals.

*Calc. phos.*—Chronic hypertrophic rhinitis in anæmic children or in association with enlarged tonsils and adenoids.

*Graphites.*—Chronic catarrh, extending to the Eustachian tubes. Tendency to atrophy.

*Hepar.*—Chronic purulent rhinitis with enlarged cervical glands. Hypersensitive to draughts. Uncovering the body brings on attacks of sneezing.

*Hydrastis.*—Simple chronic rhinitis and purulent rhinitis. Abundant muco-purulent secretion with superficial ulceration of the mucous membrane. The discharge may also be stringy and tenacious. Post nasal dropping. (*Spigelia*).

*Kali bichromicum.*—Tenacious, yellow secretion ; ulceration of the septum.

*Natrum mur.*—Simple chronic rhinitis. “ In all absence of clear indications for other drugs this is one of the best remedies where persons draw mucus from the posterior nares in the morning.”—(IVINS.)

*Pulsatilla.*—Chronic purulent rhinitis. Profuse discharge which is a bland, thick, yellow muco-pus, streaked at times with green. There is loss of taste and smell, and in order to act well there must be, according to Ivins, the typical *Pulsatilla* temperament.

*Silicea.*—Ozæna. Painful dryness of the nose ; ulceration with acrid, corroding discharge (*Merc. sol.*). Thick, fetid, post nasal discharge. Periostitis. The *Silicea* patient is pale and delicate ; predisposed to affections of the glands and bones that undergo rapid destruction ; in other words, it presents the tuberculous type. There is also nervous hyperæsthesia and tendency to neurotic affections.

#### ADENOID VEGETATIONS OF THE NASO-PHARYNX.

The muco-lymphoid glands found in the vault of the pharynx and aggregated into a tonsil-like organ known as the *tonsil of Lushka*, or the pharyngeal tonsil, are in their nor-



mal state of insufficient size to be readily detected, or to cause the least interference with free nasal respiration. Under certain conditions, however, they become much enlarged; in some instances a hypertrophy of such extent takes place that they fill up the entire naso-pharyngeal space, thus effectually preventing nasal respiration and giving rise to the pernicious habit of mouth breathing.

No definite cause can be blamed for the development of this hypertrophic condition, as it is encountered in children of all descriptions, although the so-called scrofulous diathesis, or the more pronounced glandular diathesis, *lymphatism*, are the most frequent constitutional peculiarities found associated with hypertrophied adenoids. Lymphatism is in fact interpreted as a species of constitution in which there is a tendency to hypertrophy of the lymphoid structures throughout the body, in particular the tonsils and the lymphoid structure of the naso-pharynx and also hypertrophy of the thymus glands. For this reason the two conditions often go together. Hereditary influence also offers a predisposing factor, notably tuberculosis and syphilis in the parent. The period of childhood proper furnishes the majority of cases, but infants are not exempt.

Chronic nasal catarrh; deflections of the septum; the exanthemata, and a damp, changeable climate furnish the cause, which excite the hypertrophy of these glands in children predisposed thereto.

The *pathological changes* encountered in the mucous membrane of the pharynx are an overgrowth of the mucolymphoid follicles and of the connective tissue in which they are embedded, together with increased vascularity and thickening of the mucosa. This hypertrophy leads to the formation of a large glandular mass which may attain sufficient size to entirely block up the naso-pharynx. According to the amount of connective tissue present and the mode of proliferation of the glandular elements, there will be either a soft, papillomatous growth, or a hard smooth mass, known as the individual

variety, in contradistinction to the papillomatous, which is a multiple, pear-shaped mass. The individual variety is smooth and firm, while the papillomatous is soft and irregular in contour, conveying the impression of a bunch of earth worms to the examining finger.

Adenoid vegetation belongs practically to the period of childhood, and after full maturity a physiological atrophy as a rule sets in, the pharyngeal vault being usually smooth at thirty-five, although it may be rough at as late a period of life as seventy (IVINS).

**Symptomatology.**—Chronic nasal and pharyngeal catarrh is usually associated with adenoid vegetations, especially when they have existed for a long time. While a catarrhal affection of the nose and pharynx no doubt often acts as the exciting cause of adenoid tissue proliferation, still adenoids in themselves will set up catarrh through their mechanical interference with the circulation and normal breathing. The obstruction of the naso-pharynx leads to lack of development of the frontal, sphenoidal, maxillary and ethmoidal sinuses with consequent narrowing of the face and upper jaw, which, together with the increased atmospheric pressure exerted upon the buccal surface of the palate due to lessened intranasal air-pressure and mouth breathing, leads to a gradual forcing up of the arch of the palate. This deformity results in turn in deflection of the nasal septum, on account of the upward crowding of the base of the septum. In this manner the nasal obstruction is still further augmented and hypertrophic rhinitis is invited.

Deafness from direct pressure upon the ostia of the Eustachian tubes or through an extension of the catarrhal process into the tubes is a frequent symptom accompanying adenoids.

The physiognomy is characteristic and practically pathognomonic, and taken in conjunction with the alteration in voice and deafness a positive diagnosis can be made without even instituting an examination of the posterior nares. The upper lip becomes shortened from lack of development as

a result of always having the mouth open ; the expression of the face is vacant and stupid ; the nose is pinched and undeveloped and owing to the contraction of the superior maxilla the permanent teeth become irregular in distribution.

When the condition has arrived at this stage there results as a natural consequence of the interference with the proper æration of the blood and with the general nutrition headache and mental hebetude with a certain delicacy of constitution inviting the development of neurasthenia or even serious pulmonary disease. In children who are rachitic, pronounced deformity of the chest occurs on account of the associated bronchitis and the softness of the ribs. Even in the absence of actual deformity, the "flat-chest" is frequently encountered as a result of insufficient air supply to the lungs. In several instances I have encountered cases of pulmonary tuberculosis occurring in young adults which I feel might have been prevented had the chest been properly developed. This lack of development dated back to post-nasal obstruction by adenoid vegetations which caused in turn mouth breathing ; bronchitis ; chest deformity, and ultimately phthisis. (See Fig. 39.)

Through reflex action, when in a state of irritation, adenoids in many instances bring on attacks of coughing, spasm of the glottis, and asthma. Bronchitis, due to vaso-motor paresis and irritation of the respiratory tract from mouth breathing, is one of the commoner complications of adenoids. Enuresis is a neurosis often depending upon adenoid irritation.

**Diagnosis.**—The presumptive evidence of adenoid vegetations is found in the faucies and the nasal, non-resonant voice together with the associated symptoms of mouth breathing ; naso-pharyngeal catarrh ; partial or total deafness and retarded nutrition. Naturally these symptoms are only to be encountered in well-advanced cases ; in incipient cases the age of the child and the development of the nasal obstructions, not springing from an abnormal condition of the nose

per, should always arouse a suspicion of adenoid vegetation. The positive evidence of adenoids is obtained through palpation and posterior rhinoscopy. The latter procedure is difficult, practically impossible with some children. In others, however, a very satisfactory view of the vault of the pharynx may be obtained, which is practically all that is necessary for a diagnosis, and much easier than obtaining a view of the posterior nares.



FIG. 53. METHOD OF HOLDING CHILD FOR PALPATING THE PHARYNGEAL VAULT.

Digital examination is a simple procedure and should never be neglected. Especially when deciding to operate is it necessary to gain a thorough knowledge of the size and character of the growth as well as its location in order that it may be thoroughly and intelligently removed.

The mode of procedure is the following: Press the child's left cheek against your side, encircling the head with the left arm and pressing the flesh of its left cheek in between

the teeth in order to prevent it from biting down upon the examining finger. Now introduce the index finger of the right hand into the mouth and insert it into the pharynx behind the right faucial pillar, from which position it is then brought to the median line and to the vault of the pharynx. The procedure rarely induces dyspnœa, although the child usually struggles and gags with the finger in place. The papillomatous variety convey the impression of a bunch of soft, irregular growths. The classical description found in the text-books likens it to a bunch of earthworms. In the mirror it appears as a pale, reddish-gray pendant mass, usually covered with a layer of greenish-yellow mucus.

The hard variety imparts the feeling of a smooth, rounded mass, and appears in the mirror as a pale swelling with a smooth but more or less irregular surface.

**Treatment.**—The importance of dealing with adenoid vegetations promptly on the first intimation of their presence must appeal to every practitioner who has had opportunity to see the disastrous results of the presence of these, in themselves benign growths. The condition is not to be met in a half-hearted manner, but a radical mode of procedure should be instituted from the beginning of taking the case.

Remedies have yielded most satisfactory results in many instances, but in my experience the majority of cases are amenable only to operative measures aiming at a complete removal of the growths. Especially is this so in cases of long standing, where as a rule remedies accomplish very little. It is true that at the time of puberty a physiological atrophy sets in, but the harm that has been done in childhood—the period of growth and development—is of an irreparable nature.

When a case is encountered in its incipency remedies may be tried over a period of three months, unless urgent symptoms are present, and if improvement follows and continues satisfactorily the operation may be put off or, possibly, entirely dispensed with. If, on the other hand, improvement

nly slight or absent, the sooner the operation is performed is better for the child.

*Local treatment* is difficult to carry out and its results are not satisfactory.

**Remedies.**—The remedy which has given the best results in the majority of cases is *Calc. phos.* It is generally given in the 3d decimal trituration, a grain four times daily. If indications for one of the other lime salts are present, notably, the carbonate or iodide, they should be given in preference to the phosphate.

*Arsenicum alb.* is useful for the catarrhal symptoms, especially when associated with hypertrophic rhinitis and ear symptoms.

*Sanguinaria nitrate*, 3x trit., has given excellent results. The indications are mainly clinical. "I have excellent results with this remedy and with *Calc. phos.*, the former locally and internally in the 3x trit., and the latter in the 30th or 200th, thus often avoiding operations."—(IVINS.)

Personally I give *Calc. phos.*, 3x trit., the preference over other remedies. The old school administers the *Iodide of Iron* with confidence and no doubt obtains good results; the combination of *Iron* and *Iodine* is well indicated in many instances.

*The operation* is most satisfactorily performed under a general anæsthetic, for with local anæsthesia it cannot be thoroughly done, even in children who are willing to co-operate with the physician. Ether is the safer anæsthetic, but in young children Chloroform, unless contraindicated, is preferable, on account of its quicker action and because it does not cause increased mucous secretion in the throat like ether. Profound anæsthesia is, as a rule, unnecessary.

The child being placed on the table upon its back and the shoulders elevated to let the head hang down, a mouth gag is inserted between the molar teeth on the left side. The operator now stands on the right side of the patient and introduces the index finger of the left hand into the pharynx be-

hind the soft palate in order to locate the growths. Having proceeded so far he now inserts a Casselberry post-nasal forceps, guided by the left index finger, into the vault of the pharynx and seizes a portion of the growth, which is then torn away. Piece by piece, in rapid succession, the growth is removed, after which it is advisable to introduce a Gottstein or similar curette (Fig. 54) and scrape away the remnants which may have been left.

Profuse bleeding follows, which is soon controlled by pressure with cotton mops dipped in a saturated solution of *Tannic acid* and held against the bleeding surface by means of a long curved forceps. The blood also runs freely from the nose. It should be wiped away to permit nasal respiration to set in, which usually takes place immediately after the operation.



FIG. 54.—CURETTE FOR THE REMOVAL OF ADENOID VEGETATIONS.

Both before and after the operation the nose and pharynx should be sprayed with a mild antiseptic solution (Dobell's solution; Seiler's Antiseptic Nasal Tablets), and *Aconite* administered to lessen the inflammatory reaction. Acute otitis may set in as a complication, and if antiseptic precau-

tions are not taken during the operation it may terminate in suppuration. The results of the operation are most gratifying. Nasal respiration promptly ensues (unless the case has been of long standing), the blood becomes more thoroughly aerated, with resulting improvement in the color, and the appetite and general health, and reflex disturbances are removed. Naturally cases are encountered in which an operation fails to benefit the child. Here, however, we must look for other lesions, notably, hypertrophic rhinitis and deflected nasal septum, as the cause of obstruction, either in part or in toto. Where, however, these can be excluded the results are uniformly gratifying.



## CHAPTER XVIII.

### CONSTITUTIONAL DISEASES.

#### LITHÆMIA ; URIC ACID DIATHESIS.

By the term "lithæmia" is represented a group of symptoms resulting from the presence in the blood of certain products of faulty proteid metabolism. Most prominent among these substances is uric acid, and hence the condition is frequently spoken of as the *uric acid diathesis*. Closely related to uric acid are the alloxuric bases, xanthin, hypoxanthin, guanin and adenin, and as the symptoms of lithæmia depend upon the retention of an excess of these substances in the body, the condition may be regarded as a form of auto-intoxication, to which Rachford (*American Text-book of Diseases of Children*) applies the name of *leucomain poisoning*.

Regarding the formation of uric acid in the body, Dr. Chas. Platt (private communication) writes: "There are two theories current in explanation of the origin of uric acid in the mammalian body. 1. That it is derived from the nucleinic bases, *e. g.*, xanthin, hypoxanthin, guanin and adenin; from those formed within the body and from those ingested with the food. 2. That it is derived from the amido-bodies, *e. g.*, glycocoll, leucin, tyrosin, etc.; that these are normally converted in the liver into urea; that an interruption of this normal metabolism, ureids, *e. g.*, hydantoin, allanturic acids, etc., are formed, and that these, in the kidney, are changed into uric acid. My own belief is that normally in mammals uric acid has a common origin with the nucleinic bases, viz., in the katabolism of the nucleoproteids; that it is probably not derived from the nucleinic bases, neither from those of the body nor from those of the food; that a certain percentage in health, and a larger percentage in disease, in conditions of

disturbed metabolism within the liver, arises from the glycocoll, leucin, tyrosin, aspartic acid, glutamic acid, etc., which reach the liver, after absorption from the intestinal tract, via the portal vein. A circle which may easily become vicious is established by the fact that the glycocoll itself takes origin in the decomposition of the glycocholic acid of the bile. As regards the normal formation of urea, this is, for the mammal, the end-oxidation product of proteid metabolism, intermediate steps in its formation being the ureids, alloxan, alloxanic acid, dialuric acid, parabanic acid, hydantoin, etc. A certain percentage results from the metabolism of the amido-acids in the liver, and a certain minute percentage from the uric acid carried to the liver by the portal circulation. The term 'uric acid diathesis' is indefinite, sometimes convenient, often misleading, has no significance from a chemical standpoint, and yet may not be abandoned until our knowledge becomes more definite."

It seems probable that many of the manifestations of the lithæmic diathesis are due to the xanthin bases rather than to uric acid. It is a well established fact, however, that in certain phases of the condition, known clinically as irregular gout, an insoluble urate is deposited in the tissues, causing characteristic symptoms. It may be shown, on the other hand, that many conditions, loosely designated as lithæmia, are, in reality, evidences of ptomain auto-intoxication or simply of hepatic insufficiency. Sedentary habits and over-eating will of themselves cause hepatic torpor, but it is reasonable to suppose that the descendants of gouty ancestors might be cursed with a liver that was bad from birth.

During childhood heredity plays the most important role in the etiology of lithæmia, as the other causes which may give rise to it in later life, viz., excessive proteid food, sedentary habits, alcoholism, etc., are not operative during earlier years. Prolonged illnesses frequently lead to the establishment of this condition.

*Symptomatology.*—Infants are frequently born with uratic in

arcts in the tubules of the renal pyramids ; these are washed out of the kidneys, and may be passed through the urethra or remain in the bladder, forming nuclei for vesical calculi. Older children may also have symptoms of uric acid precipitation—lumbar pains, renal colic, painful urination, hæmaturia, and, very prominently, enuresis. Examination of the urine will usually reveal the crystalline deposits.

The general symptoms of lithæmia are notably those referable to the gastro-intestinal tract, to the nervous system, and to the skin.

Nausea and vomiting in recurring attacks, accompanied by fever, acute and chronic intestinal catarrh, and stubborn dyspeptic symptoms, belong to the gastro-intestinal disturbances. Convulsions, migraine, asthma and cyclic vomiting constitute some of the most prominent nervous manifestations, while eczema is the well known cutaneous lesion of lithæmia. Disorders of vision are said to be of lithæmic origin at times. These conditions, strictly speaking, are in reality more the evidence of auto-intoxications than of gout.

Lithæmic children are as a rule delicate, dyspeptic, nervous, and excitable. They incline to be precocious and possess strong tendency to nervous and catarrhal affections. Imperfect nutrition is the keynote to an interpretation of this constitution, which belongs to that group of morbid states known as *arthritism* (BOUCHARD), in which are included the principal constitutional diseases.

The urine in lithæmia is usually scanty, high-colored, strongly acid, and deposits a large amount of uric acid and urates. Glycosuria and slight albuminuria are at times found. Lithæmic subjects frequently suffer from nephrolithiasis, and xaluria may also be present. Before an attack the urine is often passed in large quantities, being almost colorless and of low specific gravity, indicating irritation of the kidneys with insufficient elimination of solids.

While on the one hand lithæmia is frequently overlooked and the proper treatment consequently withheld,

still there is danger, on the other hand, of making a snapshot diagnosis of "uric acid diathesis" in an obscure chronic ailment. Hysteria and neurasthenia and their congeners, while they may be secondary to faulty metabolism, are in the majority of instances dependent upon hereditary defects of the nervous system, faulty education, bad home environment, or emotional causes (BARTLETT, *The Clinical Relations and Diagnosis of the Uric Acid Diathesis, Medical Era*, June, 1901). Other ailments which may be due either to uric acid or to terminal nerve irritations, such as post-nasal adenoids, phimosis and adherent clitoris, are asthma and enuresis. Auto-intoxication, or more correctly speaking, exogenic intoxication from the intestinal canal, may produce cyclic vomiting, migraine or epileptiform convulsions, but this is not lithæmia in the strict sense of the term. The xanthin bases, however, which are produced directly within the blood-stream by katabolic changes in the cell nuclein of the leucocytes, are powerful poisons and may produce important disturbances. A careful examination of the patient must therefore always be made and all other conditions excluded before lithæmia can be diagnosed positively.

**Treatment.**—The diet is of the highest importance in lithæmia, for there are many kinds of food which contain alloxuric bodies, either in the form of waste-products or as nucleoproteids, and the introduction of these into the system only adds to the burden of the already overloaded tissues. For this reason all internal organs, such as sweetbreads, kidney and liver; all meat-extracts or broths; and raw or cured meats should be absolutely forbidden. Indigestible articles, shell-fish, sweet wines and malted liquors should be withheld. Cooked meats may be allowed in moderation, but it is a fallacy to suppose that young meat is preferable to old. As a matter of fact, the contrary is true, for the flesh of young and growing animals contains more nuclein than does that of fullgrown ones. Hence beef and mutton are, theoretically, to be pre-

ferred to veal and lamb, but from the standpoint of digestibility the younger meats are more desirable. Poultry and fish are less likely to produce ill-effects than other kinds of meat. There is little difference between rare and well-cooked meats excepting one of digestibility. Sugar and very starchy foods should be given sparingly, because an excess of carbohydrates is apt to overtax the liver, which is usually impaired. The chief articles of diet should be milk, eggs, poultry, fish, oils and butter, fresh vegetables, fruit and the less starchy forms of cereal food. The patient should be encouraged to drink freely of water, preferably before or between meals, to take plenty of out-of-door exercise, and to observe regular hours for sleep.

The remedies that have proved of the greatest value in the lithæmic state in general are *Berberis*, *China*, *Lycopodium*, *Natrum mur.*, *Pulsatilla*, *Nux vomica*, *Sepia*, *Sulphur*, *Nitric acid* and *Benzoic acid*. The symptoms on which these remedies are to be prescribed are their well-known gastric, urinary and temperamental indications. (See Treatment of Renal Calculi, p. 383.) The remedies which may be called for in the special manifestations of this dyscrasia are numerous, and the reader is referred to the chapters covering these cases in their therapy.

A. C. Croftan, following a suggestion of von Noorden's, advocates the use of *Calcium* in this condition. He prescribes it in the form of the carbonate, giving ten to fifteen grain doses two or three times a day, together with a full glass of water. This mode of treatment is based on the fact that *Calcium*, on account of its affinity for phosphoric acid, combines with this substance in the blood stream, forming a phosphate which is eliminated almost entirely by way of the intestinal canal. The phosphoric acid of the blood and of the urine is thus reduced, and the sodium relatively increased; hence less mono-sodium phosphate and more di-sodium phosphate is produced, and as the latter is the normal solvent of uric acid, this substance, instead of being deposited in the tissues, remains in solution and is eliminated.

## RICKETS ; RACHITIS.

Rickets is a disease belonging exclusively to childhood, representing a pathological standstill in the normal process of ossification, with resulting softening and deformity of the entire osseous system. Associated with the lesions in the bones there is always more or less disturbance in the general health and malnutrition. The *etiology* is obscure and the course is essentially a chronic one. While some authorities, notably Kassowitz, claim that many infants show unmistakable signs of rickets at birth, still the more recent workers in this direction doubt its occurrence much before the second or third month. Personally I have encountered a few cases that presented many of the clinical manifestations of rickets apparently from birth where the mother had been in miserable health during the entire pregnancy.

*Fœtal rickets* has been described but it must be exceedingly rare. Stœltzner (*Pathologie u. Therapie der Rachitis*, 1904) states that no such condition exists, although abnormal softness of the diaphyses and swelling of the epiphyses may be observed in *osteogenesis imperfecta* and in *chondrodystrophia fœtalis*.

The majority of cases develop during the teething period. After the second year it is rare, although it may be encountered as late as from six to eight years (SCHMORL).

By far the most important etiologic factor is improper *diet*. The disease rarely develops in breast-fed infants unless lactation be prolonged beyond the normal period. In my clinic I have repeatedly demonstrated rachitic manifestations in infants from one year to fifteen months old that were still on the breast. The reason for this is the deterioration of the milk which takes place under these circumstances. Artificial feeding, however, is responsible for most cases. While a deficiency of lime salts in the food no doubt plays a prominent role in the production of rickets, as Bland Sutton demonstrated in his experiments with the lion cubs in the Lon-

don Zoo, still there are many other factors also to be taken into consideration. Clinical experience has taught us that deficiency in proteids and especially in fat, and a relatively high percentage of starch or sugar, is the usual diatetic error under which rachitis develops. Again, the improvement that takes place as soon as these percentages are properly adjusted is corroborative evidence of the close relationship of diet to the disease. The persistent use of sterilized food, notably the proprietary foods, which, at the same time, are deficient in fat and high in carbohydrates, is an etiologic factor often to be encountered.

The geographical distribution of rickets is more or less sharply defined. It is practically a disease of the temperate zone and its frequency rapidly decreases with a rise above sea-level, being quite rare in high altitudes. In large cities it is most prevalent, especially in localities with changeable and damp climate. In the cold and tropical climates it is practically unknown, and in the country districts it is rarer than in the cities. The claim is made that in some of the European cities from 80 to 90 per cent. of all children show evidence of rickets.

Unhygienic surroundings; lack of fresh air and sunshine; closely crowded quarters—these may be looked upon as contributing factors.

Zweifel's theory that rickets is primarily a form of malnutrition due to a deficient supply of lime and magnesium phosphate in the food is controverted by the lack of improvement in these cases resulting from the addition of such salts to the food. Again, that rickets is not entirely due to deficient absorption of lime salts has been proved by Ruedel, who demonstrated through urinary analyses that rachitic infants absorb and eliminate lime as well as healthy infants.

Again, the theory that certain acids, most probably lactic acid (generated in the intestinal tract), by lowering the normal alkalinity of the blood to such an extent as to interfere with the precipitation of lime salts in the cartilages, does not



seem to hold, as the alkalinity of the blood is not altered in rickets (STÆLTZNER, *loco cit.*). Although changes in the bones, similar to rickets, have been induced by the feeding and subcutaneous injection of lactic acid (HEITZMANN, BAGINSKY), still in the cases experimented upon a diet poor in lime salts was at the same time administered.

The role of heredity and of hereditary syphilis in the production of rickets can practically be ruled out, neither of them being essential to the development of the disease.

The infectious theory is advanced by Morpurgo, who cultivated a diplococcus from cases of osteomalacia in rats and by inoculating young rats with the same obtained changes in the bones bearing a strong resemblance to rickets.

According to Hagenbach rickets is due to some unknown micro-organism, his reasons for its infectious nature being (*a*), the limited geographical distribution of the disease; (*b*), its occasional epidemic appearance; (*c*), its occasional acute onset without any simultaneous change in the child's environment or food (STÆLTZNER).

Stœltzner sees in rickets a disturbance in the supra-renal glands, induced by some unknown miasm, but Wetter was unable to obtain any beneficial results from the use of supra-renal extract.

That in rickets we are dealing with a form of intoxication, there seems to be no doubt. The pathological changes in the liver and spleen, and above all, in the blood, which are often pronounced, and the accompanying nervous disturbances, offer to my mind ample evidence of such a condition. Whether this be in the nature of an infection or of an auto-intoxication of intestinal origin it is impossible to say, although the latter seems to be the most plausible.

**Pathology.**—The primary pathological disturbance in rickets appears in the periosteum. This accounts for the general sensitiveness of the body and the disinclination on the part of the child to use its extremities and its discomfort on being handled. This is followed by an irregular growth and distri-

bution of the osteogenetic cells in the centres of ossification and absorption and irregular deposit of lime salts in the bones.

The chemical composition of the bones is much altered. Thus, in the shaft of the tibia there is normally 21 per cent. water, in rickets 45 per cent. (FRIEDLEBEN). In the ribs the percentage may be raised from 44 per cent. (normal) to 66 per cent. The most important alteration, however, is the decrease in calcium phosphate. The ash (mineral constituents) may fall from 60 per cent., which is about the average in normal bone, to 30 per cent. or even lower. Such a bone can be readily bent or cut.

The first demonstrable microscopical changes take place in the periosteum, being in the nature of an abnormal proliferation of its cells. In the medullary canal, a fibro-cellular hyperplasia takes place which invades and replaces the medullary substance. The same process may affect the epiphyseal portion of the bone or even the diaphysis, leading to thickening and structural changes.

At the extremity of the long bone, where the shaft and epiphysis are joined, growth is most active, for it is by the formation of new bone from the proliferating cartilage cells and their ultimate calcification that the bone increases in length. At this point rickets shows its most marked effect upon osteogenesis. The proliferating zone of cartilage cells is increased as are also the rows of cartilage-cell columns, which at the same time lose their regular arrangement. The zone of temporary calcification encroaches upon the upper layers of the cartilage and becomes interspersed with a net-work of blood-vessels, islets of uncalcified cartilage and osteoid tissue. The arrangement of the various structures is thus greatly disturbed, this resulting from, in the first place, the irregular and hypertrophic growth of the cartilage; secondly, the invasion of the cartilage by blood-vessels, and thirdly, the irregular calcification and metaplastic changes taking place in the ossification zone.

In the medullary canal of the shaft, excessive absorption of lime salts takes place, the canal becoming abnormally large and the marrow being replaced with fibro-cellular and vascular tissue. The outer layers of the bone become thickened through excessive proliferation of the periosteum and the production of osteoid structure. In the flat bones, particularly in the occipital bone, absorption of osseous tissue in small areas results in the production of craniotabes.

These microscopic alterations in the structure of the bone explain its alteration in shape, namely the thickening of the shaft and the clubbing of the extremities, and also account for the change in the resisting power and in the consistency of the bone.

With the arrest of the rachitic process, calcification of the cartilage sets in and the bone may become abnormally hard. The hypertrophic tissue in the centres of ossification and along the epiphyseal lines is absorbed to a great extent, so that the only permanent deformity which is left, as a rule, is the distortion and bending of the bone that took place during its soft stage.

The soft structures of the body contain a normal amount of lime salts.

Pathologic changes in other organs are not characteristic and constant. The liver may be enlarged. Splenic enlargement, due to simple hyperplasia, is not uncommon. Anæmia may be pronounced. Catarrhal processes in the gastro-intestinal tract and in the lungs may be associated with rickets.

**Symptomatology.**—The characteristic deformities of the osseous system are the late symptoms of rickets; and although they are pathognomonic, their advent should not be awaited before making a diagnosis of this disease. It is unfortunate if rickets is not recognized ere marked bone lesions have developed, as the best opportunities for treatment have then slipped by.

Rickets seldom develops before the sixth month, being practically a disease of the first dentition period. Its onset

usually associated with more or less persistent diarrhœa, a moderate range of fever, fretfulness, restlessness, with tendency to kick off the covers, and local sweats. The development of anæmia, debility, profuse sweating about the head and chest, and general sensitiveness of the body to touch, indicates that the disease has become fully established. Constipation now gives place to diarrhœa, and the abdomen becomes distended and prominent.

There are cases in which the osseous changes are the first symptoms to attract attention, but they are in the minority. One of the earliest symptoms of rickets is disinclination on the part of the infant to lie on its back, as evidenced by constant rolling of the head from side to side and an effort to turn on the side. This is due to the sensitiveness of the periosteum covering the occipital bone. Associated with this is sensitiveness of the body and the local sweats, occurring mainly on the head.

The entire muscular system is in an enfeebled, undeveloped condition. This accounts for the constipation, weak heart with sluggish circulation, and the rachitic pseudo-paralysis. This latter condition results directly from the ligamentous laxity, muscular feebleness and bodily tenderness; and although some pædiatrists incline to consider these cases a form of "pressure palsy," resulting from inflammatory changes in the vertebræ producing pressure upon the cord, still such a condition, if indeed it ever exists, must be a very rare and exceptional one.

The first bony deformities to attract attention, as a rule, are the swelling of the wrists and ankles. As the disease progresses the condyles of the femurs and the ribs become involved. There is, however, no fast rule as to the sequence in the development of the deformities, and rarely are all of the characteristic lesions found in a case. The ribs become enlarged in their anterior extremity, at the junction of the rib with the costal cartilage. This deformity is described as the *rachitic rosary*, and it can be demonstrated in almost every

case on post-mortem dissection, although to find it pronounced enough to be plainly visible and palpable is by no means true in all cases. Often the epiphyseal thickening of the rib is most marked on its under surface, and we will find well developed rachitic changes at the autopsy that were not demonstrable *in vitam*. Owing to the softness of the ribs, the thorax becomes compressed laterally, with resulting projection of the sternum; this is the *pectus carinatum*, or chicken-breast. Another deformity of the chest is a groove encircling the lower portion of the thorax, the so-called Harrison's groove. This line corresponds with the lower border of the lungs and it is produced by recession of the lateral region of the soft, yielding thorax from atmospheric pressure, and the eversion of its lower border owing to the large, distended abdomen. These deformities become especially prominent as a result of diseases of the respiratory tract.

Affections of the cranial bones are among the earliest signs of rickets. Softening of the occiput, with areas of cranio-tabes, can be demonstrated, especially in the region of the lambdoidal suture. The occipital region becomes flattened as a result of the child lying on its back. The sutures are late in closing, the fontanel abnormally large, and the frontal and parietal centres of ossification are prominently thickened. These developmental peculiarities give to the head a large, square appearance, very typical of rickets. The head may also become misshapen and asymmetrical from lying more on one side than upon the other during the stage when the bones are soft and yielding.

The softness of the bones of the palate and of the jaw predisposes to the development of deformities from the act of sucking and mastication.

The spinal column suffers more or less in all cases of rickets. Owing to the softness of the vertebræ and weakness of the spinal muscles and lax ligaments, the child develops a kyphosis, when sitting up, which may result in a permanent deformity if the condition is not recognized and

corrected. Rachitic kyphosis presents a curved outline, involving the greater portion of the spinal column, and in its early stages it can be entirely reduced by laying the child upon its stomach and making traction on the column by grasping the legs (see Fig. 12). The deformity of Pott's disease is permanent, angular in outline, and involves only one or two vertebræ. Scoliosis may exist alone or in conjunction with kyphosis.



FIG. 55.—CHILD WITH RICKETS SHOWING LARGE HEAD, NARROW CHEST, PROMINENT ABDOMEN AND OSSEOUS CHANGES

The extremities suffer from bending and twisting, as a result of muscular traction or the weight of the body. The humerus and tibia suffer most frequently. Serious deformity of the pelvis rendering parturition difficult or even impossible is one of the unfortunate late results of rickets.

The eruption of the teeth is delayed and irregular, and they may decay early on account of a deficiency or irregular deposit of enamel. Rachitic teeth are typically ridged in their long axis and sometimes present a saw edge. They must not be mistaken for syphilitic teeth.

Rachitic children show a marked predisposition to a variety of ailments, referable to the nervous system, the alimentary tract, the skin and mucous membrane. Another notable peculiarity of rickets is its influence upon the course of acute illness in general—a disturbing factor, the recognition of which may prove of the greatest practical importance in the treatment of such cases.

Among the disturbances in the alimentary tract complicating rickets, chronic indigestion, chronic intestinal catarrh and obstipation are of the most common occurrence. The mucous membranes in general are prone to catarrhal inflammation, characterized by a tedious course.

The nervous system is particularly unbalanced and highly susceptible to peripheral impressions. Trifling ailments are liable to be ushered in with convulsions, and, in fact, convulsions occurring after the first year should always lead to a suspicion of rickets. Spasm of the glottis occurs almost exclusively in the rachitic. It develops after the sixth month, and continues until the second year, being intimately associated with craniotabes (ELSASSER).

The alterations in the *blood* are not constant and uniform. In all cases more or less anæmia is present, and in some, especially those with splenic tumor, there may be leucocytosis with abnormal elements (myelocytes; mast-cells and normoblasts) in the blood. Some cases almost attain to the type of a pseudo-leukæmia. The tendency is to a reversion to the embryonic type of the blood elements as the above findings indicate. The hæmaglobin and red cells are diminished in varying proportions.

The course of rickets is a chronic one, but the early institution of treatment, together with the favorable influence



fresh air and sunshine, will, as a rule, check the process promptly. After the rachitic state has become well established treatment yields slower results.

*Acute rickets* has been described and Baginsky encountered two such cases, both accompanied by high fever and terminating fatally. Autopsy confirmed the diagnosis, positively excluding scurvy. This condition, like foetal rickets, must be exceedingly rare. Stœltzner holds that it is impossible for rachitic bone changes to occur acutely.

The *differential diagnosis* rests between *hereditary syphilis*; *hydrocephalus*; *Barlow's disease* (scorbutus), and *Pott's disease*. The differentiation of the last condition has been considered above. *Barlow's disease* is a more acute disease, more frequently found in infants of the better classes as a result of exclusive feeding with proprietary foods, and is attended by swelling of the shafts of the bones, from sub-periosteal hæmorrhage, as well as joint-tenderness and swelling, besides ecchymoses in various parts of the body and hæmaturia. As this condition frequently becomes engrafted upon rachitis, its recognition may be attended with some difficulty.

Epiphyseal disease and separation is a symptom of *congenital syphilis*, which, however, occurs in the earliest months of life, other signs of syphilis being demonstrable.

*Chronic hydrocephalus* presents a head more rounded than the rachitic cranium; the face is disproportionately small in comparison with the head; the eyeballs are deflected downwards, and the mental condition is one of dullness and imbecility rather than precocity, as in rickets.

The *prognosis* is usually favorable but it cannot be estimated in an off-hand manner. An uncomplicated case without pronounced deformity of the chest, anæmia and splenic tumor is generally promptly amenable to treatment, while those with such unfavorable symptoms especially in conjunction with laryngismus stridulus present a more serious prognosis. The occurrence of pneumonia or whooping-cough in a rachitic infant is most grave. Rickets also predisposes to tuberculo-

sis, especially when there is chronic bronchitis and enlargement of the bronchial glands.

**Treatment.**—Prophylactic measures are to be directed to the mother during gestation, if rickets is hereditary in a given family. The child's diet is of the highest importance, avoiding the use of farinaceous food and giving a modified milk containing as much fat and proteids as the child can digest. It is not to be forgotten that breast-fed infants may develop rickets if weaning is not instituted at the proper time (before the end of the first year) in the case of delicate mothers whose milk is deficient in the above constituents. To early resort to solid food is to be deprecated.

Fresh air and sunshine are other absolute necessities for the infant, and the scarcity of rickets in rural districts and in the tropics is a strong proof of the prophylactic power of these elements.

In the early stages of rickets *Calc. phos.* undoubtedly stands at the head of the list of all remedies, both in the matter of frequency of indication and clinical value. The scrawny, undeveloped infant, with flabby abdomen; diarrhoeal stool containing greenish mucus, undigested casein and fat particles and organic acids; delayed teething and craniotabes, closely corresponding to the incipient period of the disease.

Later on, as the osseous changes, the anæmia, local sweating about the head, glandular enlargements and distended abdomen become prominent symptoms, *Calc. carb.* is more applicable.

The favorite old-school prescription is *Cod-liver oil* and *Phosphorus*. Of the value of the former as a food there can be no question, but I have obtained equally good results from the use of *Olive oil*, while phosphorus, when employed upon homœopathic indications, particularly those referable to the nervous system and respiratory tract, invariably yields most beneficial results. Kassowitz has been able to demonstrate that *Phosphorus* exerts a specific, selective action upon the epiphyses of the long bones, inducing an inflammation.

tory process of the bone-forming cartilage at this point thus presenting the strongest resemblance to the rachitic process. On the strength of this he was the originator of the "phosphorthérapie" in rickets, being championed by such pediatricists as Demme, Soltmann, Jacobi and others. This is certainly Homœopathy, either blindly or wilfully unrecognized. Baginsky even goes a step further and states that it does not seem to help all cases alike, but chiefly those presenting laryngismus stridulus.

*Ferrum phos.* is useful and perhaps more frequently indicated than *Phosphorus*, its well-known applicability in anæmia, bronchitis, febrile conditions and acute diarrhœas making it an indispensable remedy at some period in the disease.

Other remedies of importance are the following :

*Alumina.*—Abnormal cravings or voracious appetite ; open fontanelles ; distended abdomen ; obstipation, from inactivity of rectum.

*Bell.*—The nervous manifestations of rickets frequently call for this drug.

*Kali hydrojod.*—Preliminary symptoms of rickets. Tenderness of the entire body, but especially about the head.—(COOPER.)

*Mercurius.*—Syphilitic ancestry ; large head and open fontanelles ; offensive, oily perspiration ; glandular enlargements ; tendency to catarrhal affections with ulceration ; curvature of the bones.

*Natr. mur.*—Emaciation of the neck and thighs ; anæmia ; slight pliability of bones.

*Silica.*—Profuse sweating about the head and chest, with general sensitiveness of the body ; anæmia ; pale skin through which the bluish veins are prominently seen ; swelling of the epiphyses of the bones and affections of the cartilages in general ; skin dry and scaly, with tendency to suppurative affections, notably paronychia. *Silica* is an important remedy in rickets, standing on the same plane with the *Calcareas*, from which it must be carefully differentiated.

## INFANTILE SCURVY ; BARLOW'S DISEASE.

Scurvy is a constitutional disease resulting from faulty nutrition, the infantile type of which assumes an acute course, with a superficial resemblance to rickets, for which reason it was formerly described as *acute rickets* and *scurvy-rickets*. The disease most frequently occurs between the ages of seven and fourteen months, although it has been seen at a much earlier period, one reported case being but four weeks old. (*Amer. Pædiatric Soc. Report of Infantile Scurvy, New York Med. Record*, July, 1898.) In contradistinction to rickets, scurvy is encountered perhaps more frequently among the well-to-do than among the poorer classes, for it is among them that patent foods are more often used, the poor not being able to afford them. The immediate cause is unquestionably *diatetic*, the composition of the food being directly responsible for the development of the disease. Although it has been claimed that sterilizing or boiling the food is in itself a cause for scurvy, still there is not sufficient evidence to establish this as a fact ; and cases of scurvy developing in children thus fed depend more upon the character of the food employed than upon the manner of its preparation. It is almost invariably found that some form of proprietary food has been used either as a desiccated or a preserved preparation. In rare instances scurvy has developed in infants at the breast and in some that were taking raw milk, and, as Koplik points out, the method of preparing the food is of less importance than its composition. There must be something lacking in the food, but what this element may be we do not know. Besides the composition of the food, however, the question of its purity plays a most prominent role, and Dr. Nansen, among others, holds that scurvy is in its essential nature a form of chronic ptomaine poisoning. All of the cases that I have seen had been fed on some one of the well-known proprietary foods.

**Symptomatology.**—The characteristic symptoms of scurvy in infancy are anæmia ; sponginess and bleeding of the gums ; subperiosteal hæmorrhages, notably of the lower extremities ; general sensitiveness of the body, and pseudo-paralysis of the extremities.

The early manifestations of scurvy are a moderate amount of fever and painfulness of the extremities, most marked about the epiphyses of the bones. The child usually shows a rachitic tendency or actually has rickets, although the two conditions are entirely distinct. The gums are swollen, and may show petechial spots beneath their mucous membrane, or there is bleeding from about the teeth. The eyelids are often œdematous, but cutaneous hæmorrhages are rare.

The knee- and ankle-joints may become considerably swollen and exquisitely tender, with discoloration of the skin. Swelling of the shafts of the tibia and femur can be elicited by palpation in severe cases.

*Hæmaturia* is at times the first symptom observed, and together with tenderness of the body may be the only symptom present. Morse (*American Medicine*) recently reported several such cases, and Barlow himself recognized this fact at the time he brought the disease before the notice of the profession.

The course of scurvy is an acute one, and under proper treatment it can be shortened to a few weeks. Fatal cases have occurred, especially in those whose true nature was not recognized in time.

The *diagnosis* is not difficult when the characteristic symptoms have developed, although it can readily be confounded with *acute articular rheumatism* in the early stages. Articular rheumatism, however, is so rare in infancy that it should be the last condition thought of.

*Hæmaturia* in infancy is always to be taken as strong presumptive evidence of scurvy, and the application of the therapeutic test, namely, fresh milk and lemon juice, will soon indicate whether we have been right or wrong. The *hæmor-*

*rhagic diathesis* and *syphilis* under certain conditions may present symptoms similar to scurvy, but each of these diseases is sufficiently distinct in its clinical characteristics to be recognized as such, and here again the therapeutic test furnishes a ready means of differentiation.

**Treatment.**—In the treatment of scurvy a change of food is demanded first of all. A milk formula suitable to the child's age, preferably unsterilized, and fed in definite quantity and at regular intervals, together with the administration of fruit-juice (two or three teaspoonfuls of orange-juice twice daily), are the dietetic requirements. Fresh-meat juice can also be given at regular intervals if anæmia and prostration are marked.

Constitutional remedies are of the greatest value, particularly so when rachitic manifestations are present. The child's suffering can also be alleviated by remedies of an acute type, such as *Agave Amer.*, *Ferrum phos.*, *Bryonia*, *Rhus tox.*, *Ruta* and *Mercurius*. Deschere (*N. Amer. Jour. Hom.*, September, 1897) reports a case in which *Calc. carb.* was prescribed upon the indications of profuse perspiration about the head and neck during sleep, sour odor from the mouth, and frequent, offensive urine, with immediate improvement of the condition. For the hæmaturia, *Phosphorus* seems best adapted.

#### STATUS LYMPHATICUS ; LYMPHATISM.

The status lymphaticus presents a condition of hyperplasia of the lymphatic structures throughout the body, especially affecting the solitary follicles and lymph nodes of the intestines. The mesenteric and bronchial lymphatic glands are much enlarged and there may be associated enlarged tonsils, adenoid vegetations and enlargement of the superficial lymph nodes. The spleen is enlarged and hyperplastic. The thymus presents a notable degree of hypertrophy, and it is this condition, which is the most important lesion of lymphatism, that is looked upon by many observers as being responsible for the various symptoms belonging to the disease.

Paltauf believes that the status lymphaticus offers a lowered resistance to disease and that the sudden death is due to paralysis of the heart, while Jacobi clings to the belief that sudden death in these cases results from pressure symptoms (see p. 122). Hypoplasia of the heart and aorta has been observed in some of the cases. Blumer expresses the opinion that death results from a lymphotoxæmia, due to overproduction of the internal secretion of the thymus.

There are no symptoms characterizing the status lymphaticus aside from the tendency to sudden death and attacks of asphyxia occurring in infants. The latter were described by Opp nearly a hundred years ago as "thymic asthma." Infants frequently die in these attacks. General lymphatic enlargement, markedly hypertrophied tonsils and adenoids and swelling over the manubrium sterni may hint at the condition, but this offers no positive evidence as hyperplasia of the faucial lymphoid tissues may exist in otherwise normal children. The disease is most frequently diagnosed at the autopsy and is only suspected when an infant dies suddenly without apparent cause or when an older child, previously healthy, succumbs to some trifling operation, from the effects of an anæsthetic, or dies within the first twenty-four hours after what has appeared to be an attack of congestion of the lungs or beginning pneumonia. Convulsions are frequently associated.

#### SCROFULA; TUBERCULOUS ADENITIS.

Scrofula in its fully-developed state is a chronic tuberculosis of the lymphatics; yet the scrofulous diathesis and many of its leading clinical manifestations may be encountered in one case after case in the absence of demonstrable tuberculous lesions in any portion of the body. The general nutritive disturbance underlying the development of scrofula is responsible for the hyperplasia and the tendency to inflammatory changes in the lymphatic glands; likewise the predisposition to obstinate catarrhal conditions and to certain cutaneous



eruptions. The scrofulous diathesis invites tuberculous processes of slow and chronic type in the lymphatic glands and bones. The disease remains localized and it is more the exception than the rule for a child with tuberculous arthritis or adenitis to develop a general infection from this source.

Heubner (*Kinderheilkunde*, 1903) looks upon phlyctenular conjunctivitis as the representative type of a scrofulous inflammation and he is of the belief that it results from infection with a minimal number of tubercle bacilli—the majority of which probably undergo dissolution and so act through the agency of their toxic proteids. For this reason it is impossible to demonstrate the tubercle bacillus in the secretion from such a catarrh. The chronic, intractable nasal catarrh of scrofulous children may have its beginning in a tuberculous infection of the faucial tonsil. The frequency with which tubercle bacilli lodge in the faucial tonsils and thence get into the cervical lymphatic glands is well known.

*Secondary pyogenic infection* is another clinical manifestation of scrofula. The lesions of the mucous membrane and of the skin soon become the seat of a “vulgar” infection—some form of pus micro-organism—and this leads to ulceration or suppuration. When a tuberculous gland breaks down the necrosis is usually due to secondary infection of this nature.

In the *etiology* of scrofula, as well as in every other diathetic condition, heredity plays the most important rôle. Parents who have themselves been scrofulous, or are tuberculous, give birth to children in whom this morbid tendency is likely to become apparent. So likewise carcinoma, syphilis, parental old age, or marked difference in the ages or close blood relationship of the parents, have been looked upon as etiological factors. Acquired scrofula may result from unhygienic surroundings, especially when combined with improper feeding. The early abstraction of mother's or cow's milk from the child's dietary and a substitution of starchy foods is a potent factor in the production and fostering of scrofulous manifestations.

The division of scrofula into two types, described as the *erethetic* and the *phlegmatic*, is likely to produce confusion, as the former represents the purely tuberculous diathesis, possessing none of the features of scrofula, but a strong tendency to the development of tuberculous affections of a rapid course and extensive distribution.

*Phlegmatic Type.*—This represents typical cases of scrofula, *i. e.*, children of coarse features who are predisposed to catarrh and skin diseases showing a strong tendency to recur or to be intractable to treatment, and they are subject to glandular enlargements in various parts of the body, especially in the cervical region (tuberculous adenitis). They are usually mouth breathers and have enlarged tonsils and adenoids. The nasal catarrh produces a hyperplasia of the glandular structures and inflammatory infiltration of the connective tissue of the upper lip, causing it to become large and protruding (HENOCH). Young children are usually fat and flabby, and the abdomen is prominent. The peripheral circulation is poor.

Scrofula develops most frequently toward the end of the first dentition period. With the advent of puberty its active manifestations disappear, although in exceptional cases individuals may remain scrofulous for a much longer time.

The lymphatic glands which most frequently become infected with tubercle bacilli are the cervical, bronchial and mesenteric groups.

They become enlarged from multiplication of the cellular elements and hyperæmia, later undergoing caseation or abscess formation (secondary infection). Simple hyperplasia results in the superficial lymphatics from infection through the skin (scabies; eczema) or mucous membrane, or from the absorption of infectious material in diseases of the tonsils, ears or teeth.

Disturbances in the mucous membranes are characterized by hypersecretion, the secretion being irritating and offensive, inducing eczema and lymphadenitis in adjacent parts. Ca-

tarrhal affections are chronic in their course, and the nasopharyngeal adenoid structures, as well as the tonsils, are hypertrophied. The dangers to the scrofulous child are ophthalmia and otitis. In the former, permanent injury to the cornea may be anticipated, while in the latter life may be endangered through the advent of mastoid disease. In some instances the otitis is tuberculous in nature, but most frequently it is pyogenic.

The skin is principally attacked with impetiginous eczema; beside this, lupus, prurigo and lichen are encountered.

Affections of the joints are as a rule provoked by a traumatism, and the resulting inflammation may end in destruction of the joint. Scrofulous changes in the bones show themselves as a fungous osteitis or periostitis, the vertebræ, phalanges, the head of the femur and the lower end of the tibia being most frequently affected. These processes are purely tuberculous in nature, being invited by the peculiar vulnerability of tissue belonging to scrofula.

The *prognosis* depends upon the character of the lesions presented. Tuberculous adenitis will be separately discussed. The catarrhal and cutaneous manifestations may prove stubborn in their course. The scrofulous bone affections are serious and when suppuration takes place they often lead to death from exhaustion, amyloid disease or general tuberculous infection. The last condition is always to be feared when extensive involvement of the bones or lymphatic glands is present. Involvement of the bronchial and mesenteric glands is a very much more serious condition than involvement of the superficial lymphatics.

The *diagnosis* of scrofula rests upon a recognition of the diathesis already described and the characteristic lesions found in the lymphatic glands, the skin, mucous membranes and bones. As these localities are also affected in a specific manner by syphilis, the differential diagnosis rests between these two diseases. A careful comparison of hereditary syphilis and scrofula must, however, remove any doubt as to the na-

ture of a given case. Thus, for example, the rhinitis of syphilis develops in early infancy; it is accompanied by ulceration and destruction of the nasal septum, a result never obtained in scrofulous coryza. The symmetrical, mixed lesion of the skin, the moist condylomata and fissures of syphilis, are, again, quite distinct from the scrofulodermata, and the osseous changes appear as osteoperiostitis of the long bones (notably affecting the tibia) and osteochondritis of the epiphyses, never resulting in caries and destruction of joints, as in tuberculous osteitis.

**Adenitis.**—*Acute adenitis* is most common in infancy; tuberculous adenitis in childhood. Acute adenitis may be primary, but most frequently it complicates one of the acute diseases. The secondary form rarely undergoes suppuration excepting when it complicates scarlet fever. Primary cases are usually the result of a cryptogenic infection through the skin or buccal mucous membrane and, as a rule, break down.

**Tuberculous adenitis** may run an *acute* or *chronic course*. The acute variety may become generalized, but it is a rare form. There is another form of generalized tuberculous adenitis in which successive groups of glands become involved, the course bearing a close clinical resemblance to Hodgkin's disease.

*Local tuberculous adenitis* is the commonest variety. Its characteristic features are chronicity and its tendency to spontaneous healing. The commonest form is *cervical adenitis*, usually associated with enlarged tonsils and adenoids. It is frequently encountered in poorly nourished children and represents the typical case of scrofula. The glands may break down and suppurate, producing ugly scars in the neck, or they may undergo sclerosis or calcification. This form of tuberculosis rarely sets up a general infection and there is no direct relationship between cervical adenitis and pulmonary tuberculosis.

*Tracheo-bronchial tuberculous adenitis* is more serious than cervical adenitis, as it may result in ultimate infection of the

lungs. It is, however, frequently encountered in apparently healthy children that come to autopsy from some acute illness.

*Mesenteric tuberculous adenitis* is usually associated with tuberculous ulceration of the gut, although in some cases the latter only shows catarrhal manifestations. Its effects upon nutrition are marked and general infection or death from marasmus is the usual outcome. A moderate degree of adenitis of tuberculous origin may, however, be demonstrated in many instances in children dying of some acute illness.

**Treatment.**—Children showing a predisposition to the development of scrofula must be put under a strict anti-scrofulous regime of hygiene and diet. In the prophylaxis and moderation of scrofulous manifestations an out-of-door life, particularly a sojourn at the seashore, and a diet consisting mainly of milk, eggs, meat, and cod-liver oil, play the most important rôle.

The remedies indicated upon a constitutional basis are the *Calcareas*, *Mercury*, the *Iodides*, and such other deep-acting drugs as *Silica*, *Sulphur* and *Baryta*.

For the tuberculous lesions of the glands and bones I know of no remedy which will yield the prompt and positive results obtained from *Iodoform*. Administered in conjunction with the proper surgical treatment of such cases it hastens healing and prevents recurrence and extension of the process. The empirical use of *Iodoform* in a large number of scrofulous subjects has also demonstrated its efficacy in checking glandular swellings and preventing their breaking down; and a severe case of tuberculous osteitis of the tibia under my care, in which radical surgical measures combined with the administration of apparently well-indicated remedies (*Phosphorus*, *Silica* and *Aurum*) failed to benefit the patient in the slightest degree, was cured by *Iodoform*. Aside from its local action it seems to improve the patient's general condition, and especially increases the appetite.

The symptomatology of the following remedies demands especial consideration :

*Arsen. iod.*—Catarrhal discharges of an irritating and persistent character; debility; tuberculosis of the bronchial glands and lungs.

*Aurum met.*—Caries of the bones; foetid otorrhœa and caries of mastoid process; retarded puberty.

*Baryta carb.*—*Baryta carb.* is claimed to be related to a retardation in the development of both body and intellect. The glandular swellings are characterized by stony hardness. Beside this, enlarged tonsils and dry, scaly skin eruptions are prominent symptoms of the drug. The *Iodide of Baryta* is preferable in the throat affections of scrofulous children, although *Calc. phos.* is more useful than either of these salts in the majority of cases.

*Bell.*—Acute symptoms of a scrofulous type referable to the eyes, ears, throat, lymphatics, etc.

*Calc. carb.*—This remedy, which represents the leucophlegmatic temperament, gives us a perfect picture of scrofula in many of its phases. The catarrhal discharges from the nose, eyes and ears are offensive and irritating in character, and produce eczema and lymphadenitis in adjacent parts. The ophthalmia is very prone to become complicated by ulcerative keratitis, or phlyctenulæ make their appearance independently; the otorrhœa may lead to caries of the temporal bone, and this, in turn, bring with it the danger of a cerebral abscess. The skin is dry and generally unhealthy, eczema developing from slight irritation, while suppuration readily sets in after an injury. The lymphatics become enlarged and tend to break down. The child is fat and pot-bellied, the latter condition depending chiefly upon intestinal torpor. Other symptoms frequently observed and strongly indicative of *Calc. carb.* are craving for eggs; crusta lactea; retarded dentition; constipation with chalky stools.

*Calc. phos.* possesses more of the true tuberculous element in its symptomatology, the child being emaciated and of a less sluggish type of constitution. The stools are especially important, being loose and offensive, and containing greenish

mucus and undigested food-particles, hinting at intestinal tuberculosis. Caries of the bones has also been benefited by *Calc. phos.*, and it stands in close relationship to *Iodoform* in both its intestinal and osseous disturbances. The *Iodide* is also an important remedy in its particular sphere.

*Graphites* is one of the most useful remedies for the scrofulous skin affections, particularly when of the moist variety, with tenacious, yellowish exudations, accompanied by induration of the superficial lymphatics.

*Hepar sulph.*, together with *Mercurius* and *Silica*, is required in the suppurative conditions of scrofula.

*Silica* is indicated in caries of the vertebræ and long bones when fistulous tracts have formed (*Calc. sulph.*), and the pus is thin and offensive. The *Silica* child is pale and emaciated, the skin is thin and transparent, and the veins show through prominently. There is a strong tendency to the development of general tuberculosis, particularly when a local focus for infection is present.

*Phosphorus*.—Thin, watery pus oozing from the diseased joint; hectic fever; chronic diarrhœa; nervous temperaments. Similar to *Silica*, although the *Silica* child is more apt to be of a fair complexion, presenting the anæmia and lack of animal heat so characteristic of the drug.

*Sulphur*.—Dry, dirty, unhealthy skin; irritating catarrhal discharges; blepharitis; aversion to being washed; alternate diarrhœa and constipation; eczema and prurigo; emaciation, with voracious appetite.

**Surgical Treatment.**—Extirpation of all tuberculous glands that are accessible was widely practiced some years ago, having been warmly advocated by Sir Frederick Treves, but the present concensus of opinion seems to be that it is better to leave these glands alone unless they break down and threaten to infect the surrounding structures, when they should be incised, all necrotic tissue carefully curetted away, and the wound then packed with iodoform gauze and drained in this manner until healing takes place. This is the mode



of treatment recommended by Prof. Wm. B. Van Lennep, and is to my mind far safer and more satisfactory than extirpation. I have seen prompt recurrence, usually in deeper or more unfavorable sites, after the most careful and skillful attempts at eradication.

#### TUBERCULOSIS.

Tuberculosis in infancy and childhood presents itself in widely different clinical types; and as it may become a local as well as a general disease, it becomes necessary to describe many of its local manifestations under separate headings. Thus, tuberculous broncho-pneumonia is a condition described under the affections of the lungs; tuberculous meningitis belongs distinctively to the nervous diseases; tuberculous ulceration of the bowels to diseases of the intestines, and the glandular and osseous lesions to scrofula. Still, each one of these affections is tuberculosis pure and simple; and in order to appreciate the full meaning of tuberculous disease as it occurs in childhood, it becomes necessary to study it as an infectious disease caused by the *bacillus of Koch*.

It is noteworthy that infection almost invariably occurs in subjects presenting the *tuberculous diathesis*—a constitutional predisposition to the development of tuberculous processes of general distribution and more or less rapidly fatal course, resulting from transmitted hereditary tendencies, and reinforced by unhygienic and unfavorable surroundings. The question of diathesis is, therefore, a most important one, as its recognition offers suggestions immediately for both prophylaxis and treatment, as well as for prognosis.

The tuberculous processes invited by the scrofulous diathesis are of a localized and chronic form, quite distinct from those invited by the purely tuberculous diathesis. In the latter, tuberculous processes in the lungs, brain and intestinal tract, or a general infection, are to be anticipated. Generalized tuberculosis is the most frequent type encountered in young children, infection taking place through the respiratory tract,

the bronchial glands being attacked first, whence the process may reach every portion of the body through the channels of the lymphatics, the blood-currents, and through auto-infection from swallowing bacilli-laden expectoration. In older children its manifestations more closely approach the clinical course pursued in adults.

Primary infection through the respiratory tract is the most frequent mode of entrance of the bacilli into the system in infancy as well as in later childhood; and although primary infection may take place through the alimentary tract from an infected food supply (milk from a tuberculous cow or from a tuberculous mother), still this is by no means as common as was formerly taught. In fact, Koch in his recent address before the British Congress on Tuberculosis positively denied the possibility of the transmission of bovine tuberculosis to man. He cites the extreme rarity of primary intestinal tuberculosis in children, mentioning Biedert's 3,104 *post-mortems* with only 16 such cases; Baginsky's experience, who never found intestinal tuberculosis without simultaneous disease in the bronchial glands and lungs, and the reports from the Charite Hospital in Berlin, where in five years only ten cases were noted. Nevertheless, the necessity for sterilizing the child's food and particularly of weaning it from a tuberculous mother remains just as imperative as ever.

Even infants who present no evidence of the tuberculous diathesis or a negative tuberculous family history are in danger of being infected on exposure to a phthisical patient. There is no lack of evidence to prove that children have been directly infected with tuberculosis either by *inoculation*, as in the instance of the Hebrew infants inoculated by a phthisical rabbi through the sucking of the wound during the ritual of circumcision (LEHMANN); or by having lived in close *proximity with a consumptive*. A case bearing out the latter point is reported by Wassermann in which an infant became tuberculous after eight days' exposure. An ingenious theory against the validity of hereditary predisposition is advanced

by King (*New York Med. Record*, Oct. 12, 1901) and merits serious consideration. He believes that tuberculous parents transmit an *immunity* against the disease rather than a predisposition, and shows that children of tuberculous parents, although of frail constitution, have resisted invasion when under the same conditions children of non-tuberculous parents succumbed. In his belief, further, tuberculosis will eventually die out through natural selection and inherital immunity. He has also observed that tuberculosis in subjects with a tuberculous family history ran a slower course than in those without such a history, pointing to a partial immunity.

Cases of *congenital tuberculosis* are on record, the mode of transmission being through the blood-current. Direct transmission through the *ovum* or *spermatozoön* seems possible, but no doubt this is exceedingly rare. Jani claims to have found the bacillus in the spermatozoa. Personally, I encountered advanced tuberculous lesions in the lungs of an infant six weeks old, presenting caseation and fibrosis, strongly suggesting a congenital origin. Placental infection of the foetus has been observed a number of times. Martha Wollstein (*Archives of Pediatrics*, May, 1905) reports a case in detail in which advanced tuberculosis of the placenta was present, and the infant, which lived nineteen days, presented tuberculous lesions in the lungs and liver. The infection, judging from the histological findings, appeared to be hæmatogenous. Friedmann (*Zeitschrift für Klin. Med.*, 1901) injected an emulsion of tubercle bacilli into the vaginas of rabbits immediately after coitus and then examined the foetuses at different periods of gestation. In all cases tubercle bacilli were demonstrated and from this he concluded that the foetus may be infected directly from the father.

A *latent* form of tuberculosis is accepted by Baumgarten. In this the germs are supposed to be present in the tissues from the time of birth, not developing until favorable conditions, such as trauma or an acute illness, supervene.

The rôle of *heredity* in the etiology of tuberculosis is,

therefore, twofold. The disease may be transmitted directly, which is rare, or the child is endowed with the tuberculous diathesis, a frailty of constitution which predisposes its tissues to the ravages of the tubercle bacilli. Besides this, the child constantly breathes an atmosphere contaminated by the breath and sputum of the mother or of the several members of the household in whom the disease is active. It is, therefore, no wonder that tuberculosis was generally believed to be a hereditary disease before the true causative agent and its mode of transmission were discovered.

The large death-rate among infants from tuberculosis is appalling, the following statistics indicating the prevalence of this disease in early life: Comby (*Klinische Therap. Wochenschrift*, 1898) found tuberculosis in 12 per cent. of a series of two hundred and thirty-five autopsies upon children up to the age of two years. None of these cases were under three months, showing the relative rarity of the disease in extreme early life—a strong point against direct hereditary transmission. Osler (*Amer. Text-Book of the Diseases of Children*) quotes Miller's statistics from the Munich Pathological Institute, in which one hundred and fifty cases in five hundred were tuberculous. At the New York Infant Asylum 8 per cent. of the cases were tuberculous, and in the Babies' Hospital 14 per cent. (HOLT, *Diseases of Infancy and Childhood*.) The last statistics indicate that tuberculosis is not so frequent here as on the Continent. While not so prevalent among adults, still tuberculosis claims the greatest share of victims of all the fatal diseases that befall mankind. It has been estimated that one hundred and twenty thousand people die annually in the United States of consumption, tuberculosis of the lungs heading the list in all mortality bills.

**Pathology.**—The morbid anatomy of tuberculosis demonstrates that the tubercle is not the only lesion indicating tuberculous process. Although it is the typical product of an infection with Koch's bacillus, still another condition is

frequently observed, particularly in the lungs, where fusion of scattered areas of infection through exudative products and cell-proliferation in the alveoli result in large consolidated areas—the so-called tuberculous pneumonia. This is the form described by Laennec as *infiltrating tuberculosis*. The microscope reveals numerous non-vascular collections of cells, not aggregated into distinct nodules or tubercles, and separated from the healthy tissue by an ordinary round-cell infiltration. Again, the process may not rest with the formation of tubercles, a reactionary or secondary inflammation resulting in adjacent parts. In the lungs such an extension of the process shows itself as bronchitis and peribronchitis of the smaller tubes with catarrhal (desquamative) or fibrinous pneumonic areas.

Tubercles are divided into *gray* and *yellow*. The *gray*, or miliary tubercle, is the earliest stage, it is a grayish, translucent nodule about the size of a pin's head or millet seed, sometimes larger and of firm consistency. It consists of an aggregation of epithelioid cells surrounded by a zone of small round cells, or lymphoid elements. In the centre, multinucleated giant-cells are found. Their presence is characteristic of tubercle. The lymphoid elements are usually contained in a homogeneous fibrillated reticulum. As there is no vascular supply to the cell elements they soon break down, at first in the centre, undergoing fatty degeneration and necrosis, thus giving rise to the *yellow* tubercle.

The termination of a tuberculous process is destined to become one of degeneration through coagulation-necrosis of the cellular elements, *i. e.*, caseation. The lymphatic glands break down, and caseous pneumonic areas and cavities are formed in the lungs as the result of this necrosis. In those clinical types characterized by a preponderance of the tuberculous process in the meninges of the brain such a result is never obtained, owing to the rapidly fatal nature of the lesions. The other extreme—calcification of caseous lesions—is sometimes seen.

The virulence of the bacilli seems to exert an influence upon the nature of the lesions produced. Thus, Dr. Woodhead found that in feeding pigs with milk containing tubercle bacilli, subjected to heat, but not sufficiently high to entirely destroy the vitality of the germs, a modified form of tuberculosis was produced, in which the lymphatic glands and the joints were principally affected—a condition analogous to scrofula in children.

**Symptomatology.**—The clinical course of general tuberculosis depends upon the locality principally affected. As the bronchial glands are oftenest the seat of primary infection, the pulmonary type of the disease is the commonest encountered. In infantile cases the lungs are almost invariably involved and the bronchial glands usually show the oldest lesions. But the thoracic condition may remain unrecognized until revealed by autopsy in cases presenting a preponderance of cerebral or intestinal symptoms. Meningitis occurs next in frequency to the pulmonary form during the third year, about one-half of the cases dying with a terminal meningitis (HOLT); and although intestinal lesions are commonly found in conjunction with tuberculous disease elsewhere, they rarely exist as a primary condition. The meningitis may be secondary to a tuberculous process of the glands or bones.

Infantile tuberculosis may be divided into the *acute miliary* variety and a *sub-acute or chronic*, progressively spreading form which resembles marasmus in many of its clinical manifestations.

Children of the tuberculous diathesis show a peculiar habitus indicating a delicacy of constitution with low resisting power against tubercle invasion. This diathesis is hardly recognizable in early infancy, but it becomes especially prominent during the period of childhood proper, at which period tuberculosis runs a somewhat different course than during infancy, being more limited in its distribution and appearing as a purer clinical type. This is exemplified in the great prevalence of distinct cases of tuberculous meningitis,



tuberculous broncho-pneumonia, tabes mesenterica, tuberculous osteitis and adenitis at this age.

The diathetic characteristics are a delicate, frail appearance; small, slender bones; slight muscular development; transparent skin, through which large blue veins are prominently seen; soft, silken hair; long eyelashes; bright, languid eyes; oval face. They are of a passionate and lovable disposition, and the mind is active and precocious. In such children it requires but a slight provocation in the form of an acute illness, among which measles and whooping-cough stand most prominently, or a local catarrhal condition of the respiratory tract, to invite the outbreak of tuberculosis.

(a) The *acute miliary form* of tuberculosis runs a rapid course, terminating fatally in from three to six weeks. The starting-point of the general infection is a local focus, the most common seat of which, as above stated, being the bronchial glands. Acute miliary tuberculosis is more common in infants than in children and adults. In its early stage it is not suspected, as the child shows no characteristic symptoms excepting a febrile disturbance and perhaps a bronchial cough. In the true typhoid type the systemic infection is the most pronounced condition, local manifestations being proportionately slight in comparison to the toxæmia.

With full development of symptoms the fever runs high and assumes a remitting type, the remissions occurring in the morning hours, as a rule. The tongue is furred and dry, and the lips may become cracked and bleeding. Hydroa develop in abundance in some cases. A characteristic symptom is dyspnœa and cyanosis, and while there are usually present the physical signs of a general bronchitis, still the pulmonary condition, as determined by the physical signs, does not seem of sufficient gravity to produce the marked respiratory embarrassment. Diarrhœa, with distended abdomen, enlargement of the spleen and albuminuria are common symptoms. The urine gives the diazo reaction of Ehrlich, as in typhoid fever. The child becomes apathetic and stupid, and gradually sinks



into a state of coma and collapse, or death results from a terminal broncho-pneumonia or meningitis.

The meningeal type is preceded in its onset by characteristic nervous disturbances, such as irritable mood, constipation, cerebral vomiting, squinting and headache, and during its entire course there is a predominance of nervous phenomena. A tuberculous meningitis may run a purely clinical course throughout, or it may arise during the course of a general tuberculosis, thus only modifying the type of the disease. The same may be said of the pulmonary type, in which there is a predominance of the manifestations of an acute tuberculous broncho-pneumonia. The development of a tuberculous broncho-pneumonia is invited by the acute infectious fevers, especially when they attack children of the tuberculous diathesis. Measles, whooping-cough, influenza and typhoid fever are especially dangerous. As broncho-pneumonia so frequently complicates these fevers, the greatest care must be exercised in managing such cases. The picture is a familiar one, as it is seen but too often. An apparently broncho-pneumonia becomes tedious; the temperature remits, leading us to suspect a malarial condition or even typhoid fever; but the case continues, in spite of our best-directed efforts, towards a fatal termination.

(b) The *protracted form* of general tuberculosis, in which gradual wasting is the leading feature. This is the most common form of tuberculosis in infancy, and furnishes those lingering, emaciated cases which crowd the wards of hospitals and foundling asylums, and which are seen in the slums and dispensary clinics so numerous. The lesions are found in the lungs, spleen, liver, bronchial and mesenteric glands, intestines, kidneys and brain. In the lungs large areas of caseous pneumonia and large caseous or sclerotic tubercles may be found; the bronchial and mesenteric glands are usually much enlarged, and when cut present a caseous and broken-down interior. The spleen and kidneys and the capsule of the liver are frequently studded with miliary tubercles in

different stages of development, and the brain may show coarse tuberculous deposits on its posterior surface or a terminal basic meningitis.

The symptoms of chronic diffuse tuberculosis are those of progressive wasting, with here and there manifestations of a local disturbance due to the above-described lesions. It may develop idiopathically or follow an acute disturbance. Cough, indigestion and persistent diarrhœa may be present, or the child may be constipated and have a voracious appetite, at times manifesting promising signs of recovery, only to fail entirely sooner or later. As in the acute form, there may be a predominance of symptoms referable to the organs principally attacked; thus, a thoracic, abdominal and cerebral type can be distinguished. The tuberculous child is extremely emaciated, the chest is small and poorly developed, and the belly is large and prominent. These features, taken together with the wasted limbs, the flabby, shrunken skin, and the bright, precocious expression of the face, make up a clear-cut picture not so easily confused with other types of disease. Fever is not necessarily present, but as a rule it is present at irregular intervals during the progress of the disease, and in the terminal stage there is more or less continuous moderate elevation of temperature in association with the gradual appearance of evidence of broncho-pneumonic lesions in the lungs.

Physical examination may reveal areas of consolidation and sometimes cavities in the lungs, enlargement of the liver and spleen and of the mesenteric glands. The tuberculous processes in the lungs are not confined to the apices, as in adults, being scattered in distribution and as a rule not so extensive in area and therefore more difficult to demonstrate. There is, however, a preponderance of apical over basic lesions, and a persistent broncho-pneumonic process in the apices is always to be looked upon as tuberculous. The first râles are frequently heard in the nipple region, anteriorly (HOLT).

The disease usually terminates fatally within a few weeks

from the time that positive evidence of a tuberculous process can be demonstrated, although the premonitory stage wasting and other indefinite symptoms may go on for months before the condition shows its true character.

**Diagnosis.**—The diagnosis of the acute form is difficult in the absence of visible indications of tuberculosis, such as tuberculous adenitis and bone affections, or a clear-cut tuberculous family history. From typhoid fever it is to be distinguished by the irregular and remitting type of temperature, the absence of rose-spots, the bronchitis and respiratory embarrassment and the prolongation of the fever beyond the third week to a fatal termination. The chronic or apyretic form simulates marasmus and congenital syphilis to a degree; but here again the family history and the characteristic lesions of the skin and mucous membranes belonging to syphilis, and the old, withered look and gastro-intestinal disturbances of marasmus, with absence of fever and pulmonary symptoms, serve to distinguish these affections from tuberculosis. Where pulmonary symptoms are present and it is possible to obtain sputum for microscopic examination, the presence of the bacillus will render the diagnosis a simple matter. For the method of obtaining the sputum see the section of "Diagnosis" under "Pulmonary Tuberculosis."

**Treatment.**—The treatment of tuberculosis is hygienic, and, as far as remedial agents are concerned, nothing is to be expected from them more than to palliate and ameliorate symptoms as they arise. It is true, cases of tuberculosis of a benign type have terminated in spontaneous recovery through self-limitation of the process, and authentic cases have been controlled with well-selected remedies; nevertheless, this is an exceptional result which should never be promised or confidently expected.

Acute miliary tuberculosis is universally conceded to be beyond the pale of medical skill. A wider field of usefulness in medicines is found however, in the chronic form, in which life can be prolonged and suffering much relieved by carefully-

selected remedies, choosing from among those having a decided action upon the general nutrition, and those capable of controlling catarrhal conditions. *Iodine* and its compounds, especially the *Iodides of Arsenic, Antimony and Tin*, and *Iodoform*, unite both of these features. Besides these remedies, *Hepar sulph.*, *Calc.*, *Lycopodium*, *Sulphur*, the *Calcareas*, *Tartar emetic* and *Phosphorus* are frequently indicated for the bronchial and general constitutional symptoms. In the abdominal type of tuberculosis the *Iodide of Arsenic* is a most valuable remedy, both for the glandular swellings and the diarrhœa, and ample clinical reports are extant to verify the utility of the drug. *Iodoform* (3x to 6x trit.) has, however, given me such promising results in all tuberculous conditions, notably in the presence of glandular enlargements, and in chronic diarrhœa of greenish, watery, undigested stools that I give this remedy the preference above all others in these cases. *Calcareæ phos.* is quite similar in its symptomatology, and covers the above-mentioned condition in children of the *Calcareæ phos.* type of constitution. *Tuberculinum* has been used with apparent success by a number of physicians.

Feeding, climate and hygiene require careful consideration. An abundance of fresh air and sunshine is an absolute necessity both as a prophylactic against the development of tuberculosis and as an element in the successful care for these cases. Where circumstances permit the child should be taken to an equable and dry climate, not necessarily mountainous—the stimulating climate of the seacoast being very beneficial in cases not principally pulmonary in type. Liberal feeding is a most important element in the treatment; a highly nutritious but easily digested form of diet is to be selected, and fat administered freely to check the abnormal tissue-waste. Cod-liver oil, when it does not disagree, is the best form of fat; marrow fat, olive oil, butter and cream are desirable substitutes when called for. Infants often do best when the oil is administered by inunction. The milk formula should be adjusted to meet the infant's digestive powers, the

amount of fat and proteids being regulated according to the condition of the stools, the tolerance of the stomach and the degree of satiety obtained from the food, endeavoring to keep the percentages of both ingredients as high as is compatible with the case. Eggs, scraped beef, meat-juice and fresh vegetables, beside the various cereals and thoroughly ripened fruits, particularly grapes, are allowable in older children in the absence of gastro-intestinal disturbances.

#### RHEUMATISM; ACUTE ARTICULAR RHEUMATISM, OR RHEUMATIC FEVER.

The rheumatic diathesis is an inherited predisposition to certain forms of articular and abarticular phenomena in association with general and constitutional disturbances characterized by retardation in the process of nutrition. Although closely allied to gout and its manifestations, still rheumatism must be distinctly separated from this diathesis, to which it bears but a superficial resemblance. Its acute expressions are being recognized more and more as the result of a specific infection.

“Clinically, rheumatism may be divided into three classes: 1, articular rheumatism; 2, rheumatism of other organs, either external or internal, constituting what may be called abarticular rheumatism; 3, general, diffuse, non-circumscribed rheumatism.” (LYMAN, *Amer. Text-Book of Practice*.) To the first class belong acute and chronic articular rheumatism; to the second the rheumatic affections of the skin, vascular apparatus, muscular system, respiratory tract, digestive tract and nervous system, many of which are localized and clearly recognizable as rheumatism. The general, non-circumscribed affections present an array of symptoms referable to the internal organs and nerves which, although not so sharply defined and readily classified as the other forms, still offer strong indications of this dyscrasia. Among them, chorea and hemicrania; rheumatic neuralgia and neuritis; intestinal indigestion and chronic diarrhœa, and catarrhal

affections in general, represent the important conditions encountered.

**Etiology.**—In the etiology of rheumatism heredity stands high as the predisposing factor. Chilling of the surface by exposure to cold, by getting wet, or a sudden change of temperature, acts as an exciting cause. The actual cause for the pathological changes is still a disputed question, and the infectious, the neurotic and the lactic-acid theory have their advocates. Lactic acid, it is well known, acts as an irritant when present in excess in the blood; although the claims of Richardson in regard to the artificial production of endocarditis in dogs by the injection of lactic acid have not been substantiated (CHEADLE). The question arises, How is the excessive formation of lactic acid accounted for? Alfred Mantle considers it a ptomaine produced by the micro-organisms found in the blood of rheumatic patients; and, according to his experiments and those of Klebs, Cornil and Babes, the introduction of these micro-organisms into sterilized milk has resulted in the production of lactic acid. (COBB, *Trans. Am. Institute of Hom.*, 1897.) Viewed in this light, lactic acid is seen to be a result of rheumatic infection, and not a primary cause for the disease. The latest neurotic theory, as advanced by Bigler (*The Pathology of Rheumatism, Trans. Amer. Institute of Hom.*, 1897), defines the rheumatic diathesis as “an abnormal state of unstable equilibrium in the thermogenic mechanism, the result, in the individual or his ancestors, of too frequent or too prolonged stimulation by variations in the temperature to which they have been exposed. . . . Exposure to cold of any kind, if not continued too long, will not be followed by symptoms of rheumatism in those not predisposed; whereas, in the presence of the same cause, in the case of those whose thermogenic mechanism is in a state of unstable equilibrium, the reaction will become excessive, and the effects will first show themselves in the prodromal symptoms which characterize the onset of acute rheumatism.” He further endeavors

to show how continued reaction leads to pyrexia, excessive metabolism and excessive fibrin and lactic acid formation, which in turn cause further disturbances. The theory is learned and ingenious, but it does not explain the pathological changes of rheumatic fever, and particularly does it fail to account for the close relationship existing between rheumatism and endocarditis, the latter condition being established without all peradventure as infectious in origin. Of the lesions of rheumatism Eichhorst so timely says, "He who has seen post-mortem examinations of acute articular rheumatism is surprised how strongly the impression obtained at the corpse calls forth an infectious disease; hæmorrhages into the various organs; cloudy swelling in the heart, liver and kidneys; large, soft spleen, etc." (*Handb. der Speciellen Path. u. Therap.*) Further evidence of its infectious nature is seen in the epidemic appearance of the disease and its occurrence as a house disease, Edlefszen reporting seven hundred and twenty-eight cases to four hundred and ninety-two houses. Direct hereditary transmission is another clinical fact substantiating this view, several authentic cases being on record. As to the bacteriology, nothing conclusive has yet been decided. Mantle (*British Med. Jour.*, 1887) has demonstrated the existence of micrococci and bacilli in the blood and serous effusions, while Singer found staphylococci and streptococci in the urine. He considers the tonsils as the point of entrance of the infection, their anatomical structure and frequent inflammation as an initial symptom of rheumatic fever offering the grounds upon which this view is based. More recently he demonstrated the staphylococcus pyogenæ aureus in a case of chorea with inflammation of the elbow the condition having been preceded by follicular tonsillitis. This view, usually that rheumatism is due to a micro-organism, most likely a streptococcus which gains access to the system through the tonsils, is also advocated by Menzer in his work on the etiology of acute articular rheumatism (*Berlin*, 1902). The miasmatic origin of rheumatism is main—



tained by MacLagan (*Twentieth Century of Practice*), who likens it to a malarial infection. The relationship of scarlet fever to rheumatism is noteworthy, painful swelling of the joints, with or without endocarditis, not infrequently developing during the course of the fever. Arthritis, however, is not always rheumatism, and it is now generally recognized that there are a number of obscure conditions which fit, so to speak, into the gap between acute rheumatic fever and cryptogenic pyæmia.

The result of the investigations with the etiology of acute articular rheumatism conducted in Dr. Osler's clinic have been discouraging. Cole was unable to confirm the presence of the diplococci or streptococci found by Poynton and Payne, Meyer and others in the blood, in the cases studied at the Johns Hopkins Hospital. Osler dissents from the view that rheumatism is simply a mild pyogenic infection because rheumatic joints never suppurate and because salicylates have no effect upon such a process (*Practic of Medicine*, 1905).

**Symptomatology.**—In describing the phenomena of acute rheumatism the term *rheumatic fever* is most appropriate, as it includes both the manifestations of the articular and abarticular form of the disease. Particularly does this apply in the case of children, in whom involvement of the joints is by no means an essential requirement; in fact, absence of joint involvement is one of the peculiarities of rheumatism of children. And furthermore, I can confirm from my own experience that a severe arthritis is more frequently due to septic infection, gonorrhœa (see pages 121 and 391) or tuberculosis than to rheumatism. Arthritis confined to a single joint and of a sub-acute or chronic type is usually tuberculous. Acute *epiphysitis*, due to congenital syphilis, may be mistaken for rheumatism. Here the inflammation is confined to the lower end of the humerus, radius or tibia, the joint not being involved.

An endocarditis or pericarditis, or an acute inflammatory affection of the pharynx or respiratory tract, is at times the only indication of an attack of rheumatism. Urticaria,

erythema, fibrous tendinous nodules and chorea are likewise frequent manifestations of the rheumatic diathesis.

*Acute articular rheumatism* is usually of gradual onset, a moderate fever accompanied by tenderness and slight swelling of several joints indicating the nature of the complaint. Neither are the joints as highly inflamed and swollen as is the case in adults, nor is the fever so high and abrupt in its onset. The joints most frequently involved are the ankles, knees and wrists, but there is not that wide distribution of arthritis found later in life. The hip may be affected together with the knee, thus closely simulating the symptoms of tuberculous hip-joint disease. I have seen such cases also mistaken for appendicitis and psoas abscess, owing to the fixation of the limb, pain, and fever; here, however, the discovery of other sensitive joints and endocarditis, and a careful local examination, will readily indicate the correct diagnosis.

It is no wonder that a serious endocarditis so often gains full sway before it is suspected, when we consider how great the liability to error and how slight the indications of the true nature of the case are in so many instances. For this reason it is well to investigate carefully the ordinary colds, fevers and growing pains of children in order to determine their true nature, particularly when they occur in rheumatic families.

An attack of articular rheumatism runs a course of from two to three weeks under proper treatment and in the absence of complications. In a general way, it may be said that rheumatism shows a tendency to attack more extensively the tissues of the growing child, and to manifest itself over a longer period of time than in adults.

*Endocarditis* may exist alone as a symptom of rheumatism or accompany the articular forms, whether severe or mild. If it is discovered as a primary condition, arthritis or chorea, particularly the latter, frequently follows in its wake. Noteworthy to mention is the strong relationship supposed to exist between the development of *fibrous tendinous nodules* and

a progressive endocarditis. They were first described by Barlow and Warner. These nodules are mainly found about the joints, most commonly at the styloid process of the ulna, above the olecranon, and along the tibia and malleoli. In structure they are found to consist of fibrous tissue with an admixture of fibro-cartilage (MAYER). Notwithstanding a careful search for these nodules in every case of rheumatism and endocarditis in children coming under my observation, they have been but rarely found, and consequently were of little service in diagnosis.

*Pericarditis* is rarer than endocarditis, more commonly of the dry form, and more difficult to recognize than endocarditis. There is, however, more pain than in the latter, and, in the advent of effusion, more dyspnœa.

*Tonsillitis* represents one of the types of rheumatic inflammation, and it is a common affection of rheumatic children. The inflammation is severe; the attacks show a strong tendency to recur, and the accompanying fever depends upon the local condition. Involvement of the endocardium is possible. These are the features of all rheumatic inflammations, and beside tonsillitis the children of this diathesis are subject to *rhinitis*, *pharyngitis* and *sibilant bronchitis*, any of which may develop from apparently the slightest provocation. When endocarditis follows upon an attack of acute follicular tonsillitis it is hardly fair to refer to this as a proof of the identity of follicular tonsillitis and rheumatic infection, for in such a case the endocarditis is more probably septic or toxic than rheumatic (see p. 346).

*Muscular rheumatism* is only common in the form of *torticollis*, other groups of muscles being rarely affected in childhood.

The *cutaneous* symptoms indicating rheumatism are the various forms of erythema, urticaria and purpura rheumatica. They may occur alone or appear in connection with other local manifestations of the disease. Some chronic forms of skin disease are also dependent upon the rheumatic diathesis,

and by directing attention to diet and selecting a rheumatic remedy many intractable cases of infantile eczema are speedily relieved.

*Anæmia* is a direct result of rheumatism, and children who have been repeated sufferers from any of the above manifestations, usually exhibit a high degree of anæmia. Anæmia is especially noticeable in rheumatic fever.

*Chorea, hemicrania and gastralgia* are among the prominent nervous disturbances resulting from the action of the rheumatic poison upon the nervous system. Especially in chorea has the intimate relationship of the two conditions been so clearly demonstrated that little doubt remains as to the etiology of the majority of cases of chorea. Quite often other strong indications of rheumatism are present in these cases, among which endocarditis stands most prominently.

**Chronic Rheumatism.**—Many of the foregoing conditions are chronic in their course or lead to pathological changes of a chronic nature, yet by chronic rheumatism proper is understood the chronic articular form. It is a rare disease of childhood, resulting from an injury to a joint in the presence of a strongly-developed rheumatic diathesis, or through incomplete resolution or the products of an acute inflammatory attack. Disability from muscular contractures is also liable to occur in rheumatics, particularly after strains or other injuries to a joint. Chronic rheumatic arthritis is prone to become tuberculous. (WRIGHT.) Stengel (*Amer. Jour. Med. Sciences*, March, 1903) has found chronic rheumatism more frequently in children than in adults. It is essentially a sequel of the acute form. The joints of the fingers are commonly affected, but the lesions are not symmetrical, neither are there trophic changes in the skin covering the affected parts.

Still's disease is a variety of *arthritis deformans*, encountered in children. Together with the general enlargement of the joints there is swelling of the lymph nodes and of the spleen. The onset may be febrile. General thickening of

the soft parts is more pronounced than enlargement of the articular ends of the bones and there is no grating as in the adult form. Anæmia and wasting of the muscles is marked but there is no endocarditis. In some of the reported cases marked improvement in the condition of the joints was noted even after the disease had progressed in the usual manner for a year or two.

**Treatment.**—For those of a rheumatic inheritance much can be accomplished in the way of prophylaxis. Careful attention to the matter of clothing the child, having it wear flannel undergarments, and especially avoiding wetting of the feet and exposure in damp weather, is of the highest importance. The great danger which threatens these children is cardiac involvement. Constitutional remedies will do a great deal toward erasing the tendency to rheumatic attacks and mitigating their severity. *Benzoic acid*, *Calc. carb.*, *Lycopodium*, *Causticum*, *Kali hydrojodicum*, *Mercurius*, *Sulphur* and *Rhus tox.* are remedies of this type; they are frequently indicated upon purely constitutional symptoms, and will accomplish much in this direction.

The *diet* is of importance. Starchy and saccharine foods must be used sparingly, and fresh vegetables, young meats and fowl, milk and fat (cod-liver oil; olive oil; cream, etc.) are to constitute the main dietary. Remembering the strong tendency to anæmia, a highly-nourishing diet becomes imperative.

During acute attacks absolute rest in bed must be enforced, to save the heart and hasten the subsidence of joint-inflammation. Meat should not be permitted at this time. When considerably affected, the joints may be bathed with diluted tincture of *Hamamelis* or rubbed with chloroform liniment and wrapped in raw cotton. Hinsdale (*Medical Century*, Feb., 1902) has used an ointment consisting of one part salicylic acid in two parts lanolin with much benefit.

The following are the most frequently indicated and most useful remedies for the various manifestations of rheumatism:

*Acon.*—Fresh attacks. The early restlessness, fever and involvement of the joints is much benefited by *Aconite*, especially when the cause can be directly attributed to chilling of the body.

*Apis.*—Stinging and burning pains; cedematous swelling of affected parts and synovitis.

*Arnica.*—Intense soreness of the body; the bed feels too hard; great dread of being touched; scanty, red urine chilly when moving in bed; great internal heat and sour sweats.

*Arsenicum.*—Protracted cases. Pale swelling of affected parts; profuse sweats; great anæmia and prostration. Endocarditis and pericarditis (advanced cases; effusion, valvular insufficiency, œdema; cardiac dyspnœa, etc.).

*Belladonna* is frequently indicated for the febrile condition; general aching; sore throat; torticollis. *Phytolacca* is, however, more frequently indicated in rheumatic sore throat than *Belladonna*, and for the torticollis, *Lachnanthes* has proven useful in many cases.

*Benzoic acid.*—"Rheumatic diathesis in syphilitic or gonorrhœal patients. Urine high-colored; ammoniacal, very offensive in many diseases." (HERING.) Tearing pains as if in the bones.

*Bryonia* is one of the most useful remedies in articular and muscular rheumatism, as well as in the inflammations of the serous membranes complicating the same. In both of the latter conditions it is indicated early in the dry stage, as well as after effusion has taken place. *Rhus tox.* is frequently given when *Bryonia* is indicated, the mere symptom of restlessness leading to the choice of the former remedy. If we remember that the *Bryonia* patient may become very restless from intense pain—motion, however, giving no relief, and the restlessness being worse before midnight—we will not make the mistake of confusing these remedies.

*Calc. carb.*—Frequently indicated upon constitutional grounds.

*Cimicifuga rac.*—"Pronounced cardiac lesions, fibrous nodules, and muscular contractures due to inflammation of the tendons and muscle-sheaths." (COBB.)

*Chamomilla*.—Great irritability of temper; excruciating pains, worse at night; the child tosses about and cannot be pacified.

*China*.—Often indicated as a tonic.

*Dulcamara*.—Chronic rheumatism; marked susceptibility to changes of temperature. Also rheumatic cutaneous eruptions.

*Ferrum phos.*—*Ferrum phos.* and *Colchicin* are most effective remedies in controlling the intense pains of acute rheumatism. *Colchicin* I have found more applicable to pains distinctly located along the course of important nerve-trunks, especially the sciatic, while *Ferrum phos.* corresponds more distinctly to joint-pains, either localized or shifting about. *Ferrum phos.*

is so to speak, a cross between *Aconite* and *Bryonia* in rheumatism, its action being as prompt and certain as either of these. It must also be thought of for the *anæmia* which is liable to develop.

*Guaiacum*.—Rheumatic pharyngitis (*Phytolacca* affects the tonsils); rheumatic contractures. A useful remedy in chronic rheumatism.

*Hamamelis*.—Great soreness of affected parts, especially of the muscles. The aqueous extract, or the fluid extract diluted, has won great popular favor as a local application, superseding such lotions as potassium nitrate and laudanum, rad-water and laudanum, etc.

*Kalmia latifolia* is an important remedy when there is cardiac involvement. "Pains flitting from joint to joint with now and then a warning twang at the 'heart-string'" (HINSDALE).

*Mercurius*.—Tearing pain, not relieved by sweat; worse at night and from the warmth of the bed; joints usually swollen, with pale, puffy appearance of the same. General gastric derangement; coated tongue, showing imprints of teeth; foul



breath ; collection of saliva in mouth with bad taste ; diarrhoea. Extension to heart, lungs, pleura and meninges.

*Pulsatilla*.—Shifting pains, flying from one joint to another. The joints are highly sensitive, but usually no visible signs of inflammation are present. The child is fretful and disposed to cry, frequently changing its position in bed, which gives temporary relief. The symptoms are usually worse at night and aggravated by warmth. Gastric derangements, such as coated tongue, absence of thirst, anorexia, loss of taste or bitter taste, alternate heat and chilliness, and catarrhal affections, are usually present.

*Rhus tox.*—The pathogenesis of *Rhus toxicodendron* clearly indicates that it has a wider range of usefulness in rheumatism than any other remedy. Its selective affinity not only for the joints and fibrous tissues, but its decided action upon the respiratory tract, the nervous system, the circulatory system and the skin, stamp it as the remedy *par excellence* for any affection to which we may see fit to prefix the term “rheumatic,” in the absence of strong, specific indications for other remedies. It is true, the symptoms of *Rhus tox.* are not so markedly localized as those of *Bryonia*, *Phytolacca* or *Spigelia*, being most suitable to that class of rheumatic disturbances designated “diffuse, non-circumscribed rheumatism,” but nevertheless it may prove of use in any form, providing its leading indications are present. They are : “Drawing, tearing pains in fibrous tissues, joints, and sheaths of nerves, attended with a sense of lameness and formication in the affected parts ; with or without swelling and redness ; caused by exposure to wet, damp weather, to rain, by bathing or a strain ; WORSE *during rest and when commencing to move* ; BETTER *from continued motion and dry, warm, external applications* ; great restlessness.” (C. G. R.)

*Sulphur*.—Frequently of use as a constitutional or intercurrent remedy.

*Sodium salicylate* will certainly relieve the excruciating pains of rheumatism, but whether it materially shortens the

course of the disease or is of any value in the prevention of complications is still a matter of dispute with many leading old-school authorities.

*Cactus*, *Cimicifuga rac.*, *Colchicum*, *Digitalis* and *Spigelia* are indicated in cardiac involvement. (For the indications for these remedies see *Treatment of Endocarditis*, p. 351.)

#### HEREDITARY SYPHILIS.

In children syphilis is almost invariably an inherited disease, although it may be acquired during parturition from a primary lesion of the vulva or subsequent exposure to infection. This is usually the case when the mother acquires syphilis late in her pregnancy, for if the disease is acquired after the eighth month the child escapes direct placental infection. The term *hereditary syphilis*, strictly speaking, applies to those cases in which the ovum itself is syphilitic, either from the existence of maternal syphilis or from infection by the semen of the father—*germinal syphilis*. In such, syphilis exists from the time of conception. The foetus may acquire syphilis later through placental infection, in which case it is known as *congenital syphilis*, but the distinction is of no clinical importance. *Acquired syphilis* differs from the above forms both in the manner in which the disease gains access into the system and in the presence of the primary sore, or chancre, which is never found in inherited syphilis.

A syphilitic child may be born of an apparently healthy mother through paternal transmission of the disease, and although such a child is a menace to the community from the great degree of contagiousness of the disease, still the mother may escape infection from her own infant (*Colles' Law*). There are, however, exceptions to this rule, and mothers have been known to become infected from their own infants, showing that they were perfectly healthy while carrying a child with germinal syphilis. In the cases where the mother does not become infected from her offspring it still remains an open question whether she acquires an immunity

through the foetus or whether she is really a subject of latent syphilis. Again, a child may be born of syphilitic parents, having escaped infection, and remain immune to the acquired form of the disease throughout life (*Profeta's Law*). Until the true etiological factor in the disease shall be positively known and its biological characteristics fully understood, the subject of hereditary syphilis will be beset with more or less confusion of opinions.

Early or precocious hereditary syphilis may manifest itself *in utero*, leading to a miscarriage. Children showing active signs of syphilis at birth are seldom born alive. They may appear macerated, or the body be covered with an extensive bullous eruption. The majority of cases do not show external evidence of syphilis until several weeks after birth, but this almost invariably appears before the third month. The variety of hereditary syphilis described as *syphilis hereditaria tarda* by Fournier, in which the appearance of specific lesions is supposed to be delayed until after the third year of life, is not recognized by many syphilographers, they being of the opinion that the early manifestations in these cases were overlooked. Again, hereditary symptoms occurring in later childhood may be the result of an innocent infection (*syphilis insontium*).

The *pathological lesions* of hereditary syphilis are well developed in most of the internal organs. The lungs show an increase in the inter-alveolar connective tissue and proliferation of the alveolar epithelium (*pneumonia alba*). The liver may be enlarged as a result of round-cell infiltration of the interacinous spaces and pericellular cirrhosis; there may be gummata (rare) or simple interstitial connective tissue proliferation. These changes begin in the periportal region and spread into the acini, invading them with new connective tissue and blood-vessels.

In the bones, *epiphysitis* is a characteristic change already observed in the foetus. Other conditions will be referred to under the clinical manifestations of the disease.

**Symptomatology.**—One of the first symptoms observed in the syphilitic infant is the syphilitic rhinitis or “snuffles.” This is a dry catarrh due to infiltration of the mucous membrane and it may lead to ulceration of the septum with the production of the “saddle nose.” In severe cases the infants are emaciated and present bullous lesions on the palms of the hands and soles of the feet. This is soon followed by the development of diffuse infiltration of the skin with a tendency to scale; pustules; ulcerating lesions of the mucous membranes. In less virulent cases there appear at the end of a few weeks macular syphilides on the lower portion of the abdomen and on the buttocks; papules and pustules may co-exist. The pustules are especially common upon the face and buttocks. They have a tendency to ulcerate deeply, forming dark-colored crusts. The skin appears shrivelled, poorly nourished, and presents a brownish discoloration. Other symptoms are hoarse, plaintive cry; mucous patches in the mouth, rhagades at the angles of the mouth, anal condylomata and gastro-enteric catarrh, inducing foul-smelling diarrhoea. The syphilitic child is under-developed and anæmic; the face wears a characteristic old and anxious expression. The internal organs, as mentioned above, are the seat of diffuse interstitial hyperplasia of the connective tissue, through which destructive changes are wrought in the parenchyma of the liver, lungs, and digestive glandular system. These lesions are responsible for the malnutrition and eventual death of the syphilitic infant, although it may die with symptoms of basilar meningitis.

The *later manifestations* of syphilis, occurring in cases not malignant from the beginning, and consequently surviving, are those referable to the bones, teeth, organs of special sense and nervous system. It is readily seen how, in mild cases, slight early manifestations may be overlooked or forgotten, and how, upon the development of symptoms after the third year—even as late as puberty—the nature of the disease is not promptly recognized or suspected.

In the osseous system *epiphyseal osteochondritis* and *dactylitis* may occur early in the disease. Osteochondritis develops at the epiphyses of the long bones and by interfering with the growth of the bone may lead to deformity. The symptoms of epiphysitis are acute and simulate arthritis. The child holds the limb as if paralyzed on account of the pain. The lower end of the humerus is most frequently involved. Dactylitis presents a characteristic fusiform swelling of the fingers, also attacking the metacarpal and metatarsal bones. Ulceration often results with the destruction of the bone and integument. Hyperostosis of the tibia, resulting in rounding out of the tibial crest and curving of the shaft—the *sabre-blade deformity*—is very characteristic of hereditary syphilis. In rickets the sharp crest of the tibia remains unchanged, while deformities of the bone are most marked at its lower end. Cranial exostoses upon the frontal and parietal bones are also found in well-developed cases.

The milk teeth are delayed and decay early; the permanent teeth present pathognomonic signs first described by Jonathan Hutchinson, for which reason they are known as *Hutchinson's teeth*. The upper central incisors are dwarfed and present a notch upon their cutting surface, while other teeth show the influence of stomatitis upon their growth (see *Abnormalities of the Teeth*, p. 127).

Two other conditions to which Hutchinson has given much prominence are *interstitial keratitis* and *otorrhœa*. Otorrhœa or sudden deafness should always arouse a suspicion of syphilis. Interstitial keratitis is a frequent symptom of syphilis, developing at the time of puberty.

Nasal deformity is a characteristic sign of hereditary syphilis as well as radiating linear scars at the angles of the mouth. The latter result from ulcerating mucous patches, while the former is due to diffuse gummatous rhinitis, with accompanying ozæna.

Gummatous infiltration of the brain and cord may lead to a variety of disturbances in the nervous system. Meningitis;

epilepsy ; dementia paralytica ; tabes dorsalis and hydrocephalus are among the most important nervous affections that can at times be traced to a syphilitic origin.

As the syphilitic infant presents a characteristic old, withered look, so the older subject of hereditary syphilis may exhibit a diametrically opposite condition, namely, that of "*infantilism*" (FOURNIER). The individual appears younger, both mentally and physically, than his age would indicate.

The *diagnosis* of syphilis is not difficult in the presence of a clear family history and clean-cut consecutive manifestations of the disease, but it frequently presents the greatest difficulty when isolated symptoms are encountered. In the first place, a history of miscarriages in the mother followed by the birth of a still-born infant or one that died of "inanimation" in early infancy is strong presumptive evidence of syphilis. Secondly, the presence of snuffles at birth is an important symptom. An underdeveloped, wakeful, old- and unhappy-looking infant (in contradistinction to the bright appearance of the purely marantic infant) should always suggest syphilis and lead to a careful watch for such symptoms as hoarse cry, offensive diarrhoea, cutaneous eruptions, etc. The later manifestations of syphilis are all characteristic, and in the presence of such symptoms as Hutchinson's teeth ; radiating linear scars ; flattened nose-bridge ; dactylitis and interstitial keratitis, other symptoms are readily accounted for.

The *prognosis* of syphilis becomes the more favorable the later and the more benign the earliest manifestations of the disease have made their appearance. Death from syphilis is quite common in infants, but after the sixth month there is a good chance for the infant to survive if its nutrition can be maintained at a good standard. Probably one-half of all syphilitic-born children succumb before the sixth month. The longer life is sustained after that period, with the institution of proper treatment, the greater are the chances for ultimate recovery.

**Treatment.**—The syphilitic infant is a menace to its surroundings, for, with the exception of its mother, it is capable of infecting anyone with the disease. The lesions in the mouth and the discharges from the nose or from ulcerating papules or pustules anywhere upon the body are the sources from which infection takes place.

If a syphilitic history is obtainable, even before signs of the disease make their appearance, it is advisable to institute treatment at once.

As to remedies, there are a number beside *Mercury* which are not only frequently indicated, but which are indispensable in the treatment of hereditary syphilis. Usually, however, *Mercury* is the best remedy with which to begin the treatment of fresh cases, as it corresponds to the majority of the symptoms of secondary syphilis, the stage in which hereditary syphilis first manifests itself. When rhinitis and laryngitis are the most prominent early symptoms, inducing the so-called "snuffles" and hoarse cry, *Kali bichromicum* is indicated. So, likewise, numerous other remedies may be called for from the beginning on special indications. When using *Mercury* I have obtained the best results from the *protoiodide*, administering one to two grains of the second decimal trituration three to four times daily according to circumstances. As Bartlett well advises, the administration of *Mercury* should be stopped very shortly after the disappearance of symptoms, for there seems to be no necessity for mercurializing the infant. In the late manifestations of hereditary syphilis the *Iodide of Potash* must frequently be employed in material doses. The smallest dose which will improve the case is the proper one to employ, and I know of authentic cases in which this remedy in potency has yielded prompt, curative results. "It can frequently be well followed or replaced by the *Iodide of Calcareo* or the *Iodide of Arsenicum* in lesions of the glands; by *Silica* or *Zincum* or *Sulphur* in those of the nervous system; and by *Hepar sulphuris* or *Aurum* or *Nitric acid* in those of the osseous system." (COBB).



*Aurum*.—Tertiary manifestations; exostoses on skull, tibia and bones of forearm; dactylitis with ulceration; caries of nasal bones; defective development of genital organs; infantilism; mental depression.

*Baryta carb.*—Glandular enlargements; squamous syphilides.

*Hepar calc. sulph.*—*Hepar* has always been considered a valuable antidote to the evil effects of *Mercury*, but aside from this it is a most efficient remedy for many of the purely constitutional manifestations of syphilis. Its well-known influence over suppurative processes renders it useful in pustular skin affections and in the early stages of bone necrosis. The symptoms, "soreness of the nose on pressure, with red, inflamed eyes," hint at beginning caries of the nasal bones, and a similar condition is obtained in the bones of the skull and extremities as well. The sharp, sticking pains in the throat are similar to *Nitric acid*, but when this remedy is indicated there are other symptoms present by which a differentiation is not difficult.

*Kali bichromicum*.—Snuffles; harsh voice and hoarse cry; deep ulcers on the edge of the tongue; ulcers on the velum palati, eating through; ulceration of nasal septum (cartilaginous portion); ulcers in general, with characteristic punched-out appearance.

*Kali hydroj.*—Tertiary syphilis; diffuse and circumscribed gummatous infiltrations; mercurialization; interstitial keratitis; otorrhœa; swelling and ulcerative destruction of uvula.

*Kreosotum*.—Foul-smelling diarrhœa; the teeth turn black and crumble.

*Mercurius*.—As to the homœopathicity of *Mercury* to certain stages of syphilis, this is a fact so firmly established that it requires no further discussion. An analysis of the cases successfully treated with *Mercury* indicates that its most marked effects are the healing of ulcers and improvement in the general health, both of which belong to the truly homœopathic action of the drug (HUGHES, *Pharmacodynamics*). Its

“ tonic ” action is owing to its hæmatic power, while its control over diffuse inflammation and swelling of the mucous membranes, accompanied by ulceration and inflammations of serous membranes, periosteum and skin, depends upon its specific action upon these structures. This primary, specific action covers almost completely the early manifestations of hereditary syphilis, and the manifestations of *mercurial abuse* cover many of the destructive manifestations of the disease. Impetigo and rupia, rapid ulceration of the mucous membranes, skin and bones, etc., strongly call for *Mercury*, especially in combination with *Iodine*, as recommended above, or in larger doses when symptoms become urgent (inunctions).

*Mezereum*.—Pustular eruptions, forming thick, brownish crusts, with oozing of pus, painful at night; swelling of shafts of bones; syphilitic neuralgia.

*Nitric acid*.—Deep, irregular ulcers on border of tongue, upon tonsils and soft palate; sticking pains in ulcers; rhagades at angles of mouth; pustular and squamous syphilides; mercurial stomatitis and cachexia; urine strong, ammoniacal; condylomata.

*Sulphur*.—Syphilitic children often require an occasional dose of *Sulphur* to arouse their reactive powers or to control special symptoms. The symptomatology of this remedy is too extensive to be considered here, its sphere of action embracing both general and special indications. *Psorinum* may likewise be called for occasionally.

*Thuja*.—Flat, condylomatous lesions about the anus and ulcerating papules on the scrotum.

#### MARASMUS, OR ATHREPSIA; MALNUTRITION.

The extreme form of malnutrition in infancy leading to actual starvation is more often seen in hospitals and dispensaries than in private practice. Aside, however, from this appalling athrepsia, or marasmus, there is a large class of infants in whom the nutrition is simply below par, but whose

condition tends to become progressively worse unless active measures are taken to restore the balance of the physiological process of normal growth.

The pathogeny of infantile athrepsia is as obscure to-day as it was in 1877 when Parrot described the conditions as an independent disease following in the wake of gastro-intestinal disturbances and due to certain changes in the blood through which a reversal of the process of nutrition is effected and such pathologic processes as aphthæ, cutaneous eruptions, fatty infiltration of the liver and uric acid infarcts of the kidneys are produced.

The histological findings in the gut are by no means uniform. Baginsky insists that the mucosa is thinner than normal and that there is distinct evidence of atrophy of the intestinal tubules and villi. Heubner, on the other hand, claims that pathological changes are not constantly found and when so, that they are only the evidence of a preceding enteritis.

On the other hand, the long-continued distention of the gut with gas as a result of fermentation accompanied by the wasting of its muscular coat produces the appearance of a glandular atrophy. The careful investigations of Holt substantiate the view that there is no definite gross pathological lesion in the intestinal mucous membrane to account for the clinical manifestations.

The theory of a chronic acid intoxication of intestinal origin was advanced by Keller, who found the urine highly acid and containing an excess of ammonia. The origin of these acids lies in a deficient oxidation of the carbohydrates and particularly the fats of the ingested food. The fact, however, remains that this excessive elimination of ammonia has been found wanting in a number of cases of gastro-intestinal atrophy and has been repeatedly found in the absence of any distinct signs of wasting. In a number of my own cases the urine has been excessive in quantity and of very low specific gravity. The only abnormal chemical change noted was an increase in indican.

Arguing from the established fact that the intestinal mucosa of a marantic infant assimilates the proteids and fats of an artificial food much less satisfactorily than breast milk and consequently expends a much greater amount of glandular energy in this attempt, Heubner explains the failing nutrition on the grounds of a disturbed balance of energy, in other words, waste of energy on the part of the organism.

My personal investigation of the gastric contents of cases of marasmus (*Hahnemannian Monthly*, May, 1903) has shown that in a well developed case there is a total absence of free HCl. and that the amount of free hydrochloric acid in less pronounced cases bears a definite relationship to the prognosis. Indeed, where the emaciation is the result of some other disease, such as tuberculosis, I found more or less free acid, while in genuine marasmus it was absent. I recall a case of marked wasting as a result of ileocolitis seen with Prof. Bartlett. We found the HCl. but slightly reduced and a good prognosis was given. The child promptly recovered under careful dieting.

The *etiology* of marasmus is not always clear. In some infants there is undoubtedly a congenital feebleness of constitution which renders them incapable of conquering in the struggle for existence. Here heredity is an important factor, and we may find evidence of constitutional disease in the parents; on the other hand, they may be perfectly healthy. Extreme youth of the mother, and frequent pregnancy at short intervals is often noted on the maternal side of the history. The surroundings play an important role. Crowded quarters and lack of fresh air and sunshine are strong contributing factors. The ordinary hospital ward is a most undesirable quarter for infants convalescing from an acute illness and unless promptly removed therefrom they soon show signs of failing nutrition. Some believe that infection of one infant from another, possibly through contaminated food, may take place, although there is no proof that specific bacteria play a part in the etiology.

**Symptomatology.**—The infant may be delicate at birth, have difficulty in digesting its food even when breast-fed, and its progress follow a weight curve that is marked by progressive loss of weight interrupted by periods of temporary gain or standstill. More frequently the infant appears normal at birth and gets on perhaps as well as the average case up to from the third to sixth month, when as the result of some acute illness or what is more common, a change in the food, the nutrition gradually goes wrong. It is by no means necessary that the infant should have been on breast milk and that a change to artificial feeding be instituted in order to bring about this condition. A sudden change during artificial feeding to an ill-selected diet or the more gradual ill-effects from a diet that is unsuitable or insufficiently nourishing will accomplish the same results, especially when the environment is such as to favor marasmus. The emaciation progresses until the infant is reduced literally to skin and bones. The face has an old, wrinkled appearance, the eyes being sunken and the small triangular chin showing in marked contrast to the large head; the chest is small and the ribs are plainly visible while the abdomen is large and distended. Through the thin abdominal wall the stomach and coils of dilated intestines can often be seen. The skin is pale and transparent. There is more or less intertrigo about the genitals and buttox and a few scattered boils are not uncommon. Anæmia is marked. The child presents the picture of distress and restless anxiety.

On account of the adynamia these infants are inclined to develop œdematous swelling of the face and extremities, which comes and goes. A temporary gain in weight may result from this œdema. The urine is normal under these circumstances. The temperature runs a subnormal course. An occasional rise to 99° or 100° in the rectum occurs when acute indigestion intervenes, but this is only transitory. I have seen it running between 96° and 97° F. in the morning (rectal) for weeks with ultimate recovery.

The *stools* vary in character. To all appearances they may be normal, excepting for an increased acidity. They tend to vary from day to day in number, color and consistency. Usually they are large and contain light colored curds with greenish mucus. Alternate constipation and diarrhœa is frequently seen.

The appetite is variable. Sometimes for a considerable period it is voracious and the child does not seem to get satisfied. Then, again, it may be lost and there may be difficulty



FIG. 56. —INFANT ONE YEAR OLD WITH MARASMUS

in inducing the infant to take sufficient nourishment. In some instances **acute inanition** results from the refusal on the part of the infant to take its bottle. On account of the weak digestion and fermentation, colic is frequent and considerably complicates matters.

The *duration* is difficult to foretell. The child may die suddenly from an intercurrent diarrhœa or broncho-pneumonia; gradual and persistent improvement may follow proper treatment or the case may drag on with exacerbations and ameliorations far into the second year.

The *prognosis* is always grave, but it depends much upon the care the child can receive. Thousands of cases that die annually could be saved if they could be removed to more favorable surroundings and receive more skillful and conscientious nursing. It is marvelous what persistent watching and self-sacrifice on the part of the mother or nurse will accomplish in some cases with apparently the least hopeful outlook.

**Diagnosis.**—The differentiation between marasmus and *tuberculosis* is not always easy. It is said that the tuberculous infant is bright in appearance and not so prostrated and apathetic as the marantic infant, but this is not a reliable sign. In tuberculosis we have continued fever as a more or less constant symptom; at any rate, there will be distinct febrile movements at some time or another during the course of this disease. Besides, repeated careful examinations of the chest will ultimately reveal evidence of tuberculosis and we may also be able to detect enlarged mesenteric glands by palpation of the abdomen. Persistent diarrhoea with pus in the stools and at times blood speaks strongly for tuberculosis.

**Malnutrition** is a much commoner condition than marasmus. It may be the result of premature or inherited feebleness of constitution, or follow after some acute illness, notably a gastro-intestinal affection. Again, malnutrition is a prominent symptom in tuberculosis, syphilis and severe rickets.

Its most usual cause is improper feeding and unhygienic surroundings. As to the last named factors, they are just as likely to be encountered in well-to-do families as among the poorer classes, for here proprietary foods and close, overheated nurseries come into play. In older children anæmia and malnutrition often date back to an attack of one of the infectious diseases or result from improper eating and school-hygiene. The *diagnosis* of simple malnutrition rests upon the exclusion of an organic disease or infection of which it might be only symptomatic.

**Treatment.**—The regular weekly weighing of the infant is an absolute necessity and the only accurate guide by which



we can judge of the progress of the case. The evening and morning temperature should be taken regularly, as this will indicate to us whether or not we must resort to artificial heat or extra clothing; also whether the infant must be kept in bed or taken out in the fresh air. With a persistently sub-normal rectal temperature I have found it best to keep the child in bed, well clothed and a hot water bag at the feet. Such children should not be bathed but gently washed and then rubbed with warm olive oil. Very young infants who are too much exhausted by dressing and undressing can be wrapped in raw cotton.

Of the highest importance is the *diet*. If the infant be breast fed we must determine by examination of the milk whether it be sufficient in amount and of proper chemical composition. If the milk be at fault and appropriate treatment applied to the mother does not improve the same, we must try a wet nurse. If the milk is simply deficient in quantity, mixed feeding should be instituted.

As it is not always possible to obtain a wet nurse, we should bear in mind that in modifying the milk for a delicate or marantic infant it must be of a strength that would be suitable for a much younger infant than the one in question.

It is generally held that the proteids are the elements of the food that cause all the trouble in feeble digestion and there has been a tendency to cut them down to almost nothing while the fats are administered liberally. This is the mode of practice that my clinical experience has taught me to be erroneous. Some years ago I learned that infants who could not take milk, even when highly diluted, could often take it in fairly strong proportions if all the fat were removed. This is not true in every case, but there is a large class of infants who digest fat less satisfactorily than proteids and *vice versa*. Some time ago a colleague consulted me concerning a case under his care, an apparently healthy infant of eight months, that would not gain weight, although the milk seemed to be properly modified. There were some signs of

gastric indigestion and I advised him to take the cream out of the food entirely. A month later he told me that the child began to gain immediately, but every time he tried to go back to the cream, the gain ceased. Holt has recently reported several cases in which serious toxic symptoms resulted from giving too much cream—the usual reason for giving so much fat being to overcome constipation. Edsall reported similar but less acute disturbances in older children, and here he demonstrated the presence of the lower fatty acids in the urine.

The element of the food that is most easily assimilated and that is most required in these cases to maintain the body heat and keep the machinery going is the sugar, or carbohydrate. That is why condensed milk, which contains a low fat and proteid percentage and a high carbohydrate percentage often agrees after the physician has racked his brain in the attempt to find a suitable milk-formula. It is eminently better, however, to apply this principle in modifying the milk than to have the infant put on such an inferior article.

Milk sugar is preferable to cane sugar in these cases for several reasons. In the first place, it is more easily assimilated, and can be given in larger quantities. Secondly, it does not so readily undergo fermentation in the intestinal tract, but when there is a tendency to diarrhœa it may aggravate this condition. Cane sugar and even starch should not be depended upon as a food in early infancy. Cane sugar may produce untoward effects in certain infants, such as gastric irritability, vomiting and colic. I have seen cases in which every attempt to substitute granulated sugar for lactose was followed by vomiting.

The chief function of starch in early infancy is to render the casein of the milk more easy of digestion. This is purely a mechanical effect. For this purpose we dilute the milk with barley-water. When milk is not borne well it is a good plan to interpolate several bottles of mutton broth made with rice or barley in the feeding schedule.

I have not had happy results from the predigestion of starch

solutions with malt diastase. On the other hand, dextrinized starch is well born in many instances. Baked flour, or a water-cracker rolled into a powder and then boiled with sufficient water to make a thin pap and a little milk and sugar added is well borne by infants of a year or older.

For the class of infants who do not digest the proteids of milk well, Edsall has suggested bean flour, on account of its high proteid percentage. He used it in a number of marantic cases in a solution that was subsequently dextrinized, and reports good results. Dr. S. W. Sappington experimented with this food at the Children's Homœopathic Hospital, but his results were not encouraging. The use of peptonized milk does not give the results expected of it. It is not so much faulty digestion as faulty assimilation that really lies at the bottom of the trouble. The good results obtained from Peptogenic Milk Powder are, to my mind, due more to the milk sugar and bicarbonate of soda it contains than to the pancreatic extract.

*Stimulation* is at times called for. A few drops of brandy, well diluted, given during periods of great depression, has seemed helpful. Panopetone may also be tried.

On account of the anæmia, freshly prepared beef juice (diluted) should be given in small quantities daily (ʒss to ʒj). Diarrhœa would temporarily contraindicate its use. We know that even human milk contains insufficient iron to supply the requirements of the organism after a certain period, as has been pointed out by Bunge, and that the infant actually draws from the store of iron present in its tissues at birth to sustain the hæmoglobin percentage of the blood. Consequently anæmia develops if milk is continued as the sole food beyond a certain time, and more markedly in subnormal than in normal infants.

Instead of giving the usual quantity of food, it may be necessary to use a smaller amount at shorter intervals before the digestive tract will tolerate even a weak milk mixture. This, like every other question with the cases, must be ascertained by trial and experimentation.

The question of the use of *alkalies* in the food often arises. When there is vomiting of curds or the passage of curds in the stools, sodium carbonate should be added to the milk in small quantities (2 to 3 grs. to the bottle). This will prevent the formation of the tough curds of paracasein chlorid and allow the more delicate curds of casein to enter the intestinal tract where they will be digested by the pancreatic juice. If there are loose, acid stools and much gas, lime water is preferable. I have occasionally seen beneficial results from the administration of a few drops of dilute hydrochloric acid in water, half an hour after nursing, where there was a deficiency of the gastric secretion. The bicarbonate of soda, aside from its action upon the casein, also appears to exert some influence over the *acid intoxication* that plays so important a role in many of these cases.

Orange juice, on account of its beneficial effects in rickets and scurvy, may be used with advantage, especially when there is constipation and when the infant has been taking sterilized milk for some time. When the stools become highly acid and irritating the carbohydrates must be cut down and proteids (egg albumin, meat broth) increased, while in offensive and alkaline stools the carbohydrates must be increased and proteids cut down.

In looking over the list of remedies recommended in depraved states of nutrition, the deep acting constitutional ones stand in the foreground. Much benefit is derived, however, from paying attention to the acute symptoms as they arise and prescribing such remedies as *Nux vomica*, *Podophyllum*, *China*, etc., intercurrently.

The *calcareas* seem indicated in the majority of cases, especially *Calc. phos.* *Iodine* is strongly related to emaciation and glandular atrophy, and the *iodides* are often indicated, especially the *Iodide of Arsenic*, when there is great prostration, nervous irritability and restlessness; tendency to diarrhoea; dropsical swelling of the face and extremities.

*Sulphur* has many of the symptoms of marasmus, and it

suits especially the cases with cutaneous eruptions ; intertrigo ; irritating stools and urine. *Mercurius* naturally suggests itself where there is a suspicion of syphilis.

*Lycopodium* and *Natrum muriaticum* are important in malnutrition and emaciation, and will suggest themselves by their characteristic symptoms.

## CHAPTER XIX.

### ACUTE INFECTIOUS DISEASES.

#### EXANTHEMATA.

The exanthemata constitute a group of acute infectious fevers belonging to the period of childhood, occurring epidemically, and characterized by the eruption of an exanthem upon the surface of the body. To this class belong *measles*, *rubella* and *scarlet fever*. Although a specific causative micro-organism has not yet been demonstrated in any one of these diseases, still there is no doubt as to their infectiousness, and it is quite likely, as Welch (*American Text-Book of Practice*) states, that they depend upon another form of micro-organism, not a bacterium, for the demonstration and study of which we are at present not fully equipped. In the light of the most recent investigations it appears that a number of the infectious diseases of unknown origin are due to a protozoon and not to a bacterium.

#### MEASLES, RUBEOLA.

Measles is one of the commonest of all acute diseases of childhood and there appears to be a universal susceptibility to the disease as few people go through life without having had it either in childhood or in later life. A child that has been exposed to measles rarely escapes contracting the same. It occurs preferably in epidemics during those months favoring catarrhal affections; spring epidemics are usually the severest. One attack affords immunity against another. The period of incubation is from ten days to two weeks in the average of cases. Contagiousness is present from the time of invasion, being most pronounced at the height of the catarrhal manifestations and fever. It rapidly van-

ishes with the disappearance of the eruption, and at the end of the third week there remains little or no danger of contagion. The contagion is usually spread by close contact, and is seldom conveyed by means of intermediate objects or a third person, it also being readily destroyed by thorough airing and fumigation. Measles, however, is more readily disseminated than scarlet fever or diphtheria and an epidemic is more likely to attain wide-spread proportions than in the latter diseases.

**Symptomatology.**—The course of a typical case of measles is in three stages. These are characteristic to the exanthemata in general, but most clearly defined in measles. They are: the *stadium prodromorum*, or *prodromal stage*; the *stadium eruptionis*, or *stage of eruption*, and the *stadium florescentiæ*, or *stage of desquamation*.

The first stage is characterized by fever and catarrhal symptoms of gradual onset, showing themselves as a cold in the head, with bloodshot eyes and lachrymation, accompanied by chilliness and headache. The catarrhal process extends to the larynx and trachea, resulting in the characteristic hoarse cough. On the third day single, lentil-sized red spots are seen upon the roof of the mouth and soft palate, frequently being observed twenty-four hours before the eruption upon the skin makes its appearance. Koplik's sign appears even earlier and is more truly pathognomonic of measles in the period of invasion. He describes this buccal enanthem as follows: "If we look into the mouth at this period we see in a strong light the usual redness of the fauces, perhaps not in all cases a few red spots on the soft palate. On the mucous membrane lining the cheeks and lips (buccal mucous membrane) we see a distinct and pathognomonic eruption. This consists of small irregular spots of a bright-red color; in the centre of each spot is the interesting sign to which I wish to call attention. In strong daylight we see a most minute bluish-white speck. These minute bluish-white specks in the centre of a reddish spot are absolutely pathog-



nomonic of beginning measles"\* (*N. Y. Med. Record*, April 9, 1898). This sign is present in all cases twenty-four hours before the skin eruption, and often three days preceding it. (KOPLIK.)

The second stage begins on the fourth or fifth day. The eruption makes its appearance first on the face in the majority of cases, accompanied by increased fever. Thence it spreads over the entire body surface, the eruption being completed in two to three days. Its spread, however, may be irregular and interrupted, and desquamation may occur on one portion of the body while the eruption is appearing on another. The exanthem is the product of a superficial dermatitis, with papule formation through round-cell infiltration about the papillæ, the cutaneous glands and small blood-vessels. There may be also œdema of the skin accompanying the inflammatory process; this is most prominently seen upon the face. The eruption proper consists of numerous, roundish, lentil-sized red spots, slightly raised above the level of the surrounding skin, or containing in their centre a little papule. Where they are very numerous they coalesce, forming crescentic plaques, or they may fuse entirely into large, spotted areas (*morbilli confluentes*). Cases in which the hyperæmia is so great as to cause cutaneous hæmorrhages are described as *morbilli petechialis* or *black measles*; in these cases the eruption assumes a dark color from petechial hæmorrhages. Petechial measles is by no means always a more serious condition than the ordinary form; in fact, I have encountered a number of cases running a rather mild course, in which the eruption assumed this hæmorrhagic type.

A distinctive difference between the eruption of measles and that of scarlet fever is its behavior to point-pressure: "The spots disappear by finger-pressure, but the redness soon reappears from the centre toward the periphery" (HARTMANN, *Die Kinderkrankh.*, Leipzig, 1852) in measles, while in scar-

\* The first article upon this subject appeared in *Archives of Pediatrics* December, 1896.

let fever the redness reappears from the periphery toward the centre. Dr. Hartmann, however, offered no explanation for this phenomenon, which I think is easily understood from a close study of the eruption. In measles we have papules surrounded by areas of erythema, and by applying firm pressure to a patch of eruption with the finger-point we force the blood from the erythematous area surrounding the papule, but do not completely deplete the hyperæmic papillæ forming the papule, which recovers itself quickly through its great vascularity, for which reason the redness seems to reappear or even persist in the centre of the compressed skin area. In scarlet fever, on the other hand we have either a diffuse hyperæmia or a fine, closely-aggregated miliary eruption, which behaves like the erythema surrounding the measles papule; in other words, the area of skin pressed upon is completely depleted, there being no central papule, and the redness reappears from the periphery toward the centre, as the greatest amount of pressure has been brought to bear upon the centre of the area, and consequently the greatest amount of depletion.

In young children convulsions sometimes occur at the time the eruption makes its appearance. The catarrhal symptoms reach their acme, and broncho-pneumonia and troublesome diarrhoea are to be feared during this period. Catarrhal inflammation of the conjunctiva, nose, pharynx, larynx, trachea and bronchi are so closely associated with the course of an attack of measles that they are really to be looked upon as characteristic lesions of the disease. The strong tendency for the process to extend from the bronchi into the bronchioles and air-vesicles is one of the most dangerous features of measles, and almost every fatal case is directly due to pneumonia or exhibits signs of the disease.

The inflammation of the pharynx and larynx may become croupous, and suppurative otitis media may appear as a complication at this stage, although neither of these conditions are as common to measles as to scarlet fever.

In the alimentary tract a similar catarrhal condition may

become established, showing itself as anorexia, vomiting, heavily-coated tongue with enlarged marginal papillæ, and diarrhœa. The latter, when once established, is liable to continue throughout convalescence.

At the end of about four days the eruption begins to fade, disappearing first in those localities where it was primarily seen. In mild cases it has already become much paler at the end of twenty-four hours, and it may disappear entirely from one part while another part is being invaded. With the fading of the rash *desquamation* takes place in the nature of fine, branny scales, first noticed upon the face and neck. It is completed in a week in the average case, seldom continuing for a much longer period.

The eruptive period is prolonged in those cases in which it becomes hæmorrhagic. Here it assumes a deep-red color, gradually becoming darker (ecchymotic) and slowly fading out as the blood-pigment is absorbed. Again, the eruption may suddenly disappear, indicating great adynamia and heart failure. The characteristic "measly odor" is most prominent at this time, although it begins to develop during the height of the fever and catarrhal manifestations.

The temperature is not high in mild cases, being highest during the eruptive period, when it may reach  $104^{\circ}$  F. for a short time. In the average case there is an abrupt rise at the point of invasion—about  $102.5^{\circ}$  F. (initial fever). It soon falls to a lower period, not rising again until the fourth or fifth day, when the eruption makes its appearance. At this stage it may reach  $104^{\circ}$  F. and higher. In a day or two

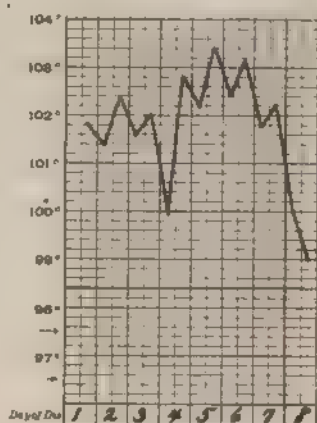


FIG. 57.—TEMPERATURE CHART FROM A CASE OF MEASLES.

it drops by crisis, unless it is sustained by a complicating broncho-pneumonia, etc.

Among the many *complications* liable to arise during the course of measles or appear as sequelæ, the following are the most important and most frequent in occurrence: Broncho-pneumonia (children under three years); lobar pneumonia, pleuro-pneumonia and empyema (three years and over); membranous croup; putrid sore throat; noma; entero-colitis; conjunctivitis and keratitis; otitis media.

The frequency with which tuberculosis develops after measles is noteworthy. In some instances latent scrofulous lesions are stirred up by the attack, while in others it appears that primary infection occurs directly upon the subacute pneumonic process lingering after convalescence. The congestion of the bronchial glands which accompanies measles renders them more liable to infection with the tubercle bacillus. According to Osler, tuberculosis is the most important sequela—either an involvement of the bronchial glands, a miliary tuberculosis, or a tuberculous broncho-pneumonia. Homœopathic authorities are, however, not inclined to take such a grave view in cases of measles under homœopathic treatment.

The *blood* in measles shows a trifling degree of anæmia and instead of leucocytosis there is an actual leucopenia in uncomplicated cases (COMBE). The urine may give the diazo-reaction, but albuminuria is rare.

**Treatment.**—The child should be put to bed in a well-ventilated, moderately-darkened room as soon as the disease is suspected, maintaining a temperature of 65° F. when possible. It is unnecessary to render the room dark and cheerless, an effectual shielding of the eyes from direct bright light being all-sufficient. The child should be kept in bed until every trace of the rash has disappeared, which usually takes place about five or six days after its first appearance. The removal of the branny scales of epidermis is greatly facilitated by rubbing the child with olive oil, followed by a

sponging with tepid water and Castile soap. This measure should be employed for several evenings in succession after the febrile symptoms have abated. During the febrile period there is no objection to the cleansing sponge-bath of tepid water. If conjunctivitis be present the eyes should be flushed several times daily with a 2 per cent. Boric-acid solution.

In cases in which the rash is tardy in coming out, or in which there is a recession of the same, a warm bath or pack is of great service. With recession of the rash the condition often becomes grave. When due to cardiac failure stimulation is indicated, and a hot-mustard bath is a valuable adjuvant when serious congestion of internal organs (broncho-pneumonia, meningitis, etc.) exists as a complication.

In dieting cases of measles we must bear in mind the tendency to diarrhoeal conditions, just as in scarlet fever we must anticipate nephritis.

During convalescence the diet should be highly nutritious, consisting largely of milk, eggs, fresh vegetables, lamb-chops etc. If a tendency to tuberculosis exists, cod-liver oil may be added with advantage. A week should elapse before the child is permitted to leave the house, and by the end of the third week from the commencement of the disease he may be allowed to commingle with other children, as the infectious period has passed over by that time.

The following remedies will be found to cover the usual cases :

*Aconite* corresponds to all of the early symptoms of the average cases of measles, and when given in time will so control the disease that it frequently becomes unnecessary to give any other remedy during its entire course. It is hardly necessary here to give its indications. In infants, however, when the fever is high and nervous symptoms are prominent, I more frequently find *Belladonna* useful.

*Apis*. —Confluent eruption, with pronounced œdema of the skin ; œdematous swelling of the throat , cerebral complications.

*Arsenicum* is indicated in those adynamic cases in which there is pronounced prostration; scanty rash; anxiety and restlessness; pneumonia.

*Bryonia*.—Cases calling for *Bryonia* are characterized by a predominance of catarrhal symptoms from the very beginning with tendency to extend to the finer bronchial tubes and involve the pulmonary parenchyma. The rash is slow in coming out, but, when once established, it is usually abundant and characteristic. The accompanying symptoms are dry, painful cough; great lassitude and irritability; anorexia, with thirst for large quantities of water; constipation, etc. *Bryonia* is looked upon somewhat as a specific to bring out the rash, but any well-selected remedy will accomplish the same result, notably *Pulsatilla* and *Gelsemium*.

*Camphora*.—"In those dangerous cases where the face grows pale and the skin cold, assuming a bluish, purple color, with utter prostration and spasmodic stiffness of the body." (C. G. R.)

*Coffea* is a valuable remedy for the short, dry, teasing cough of measles, frequently becoming a most distressing complaint in nervous, delicate children.

*Euphrasia*.—Profuse corroding discharge from the eyes, with profuse, bland, nasal discharge (*Allium cepa* has the opposite condition).

*Gelsemium*.—"After *Aconite*, great deal of coryza; drowsy, with fever heat; no thirst. When the eruption turns livid, with cerebral symptoms" (C. G. R.).

*Kali bichromicum* is indicated in measles when there is a deep, loud cough, with expectoration of stringy, yellowish mucus; intense conjunctivitis, sometimes going on to keratitis and ulceration; stitches in the ears, extending into the head and neck; watery diarrhœa, with tenesmus; ulcerated sore throat. Even when the symptoms are not so severe or characteristic as above stated, this remedy is frequently of great value, especially when *Bryonia* does not control the bronchitis as promptly as it should. It is followed well by *Pulsatilla*.

*Lachesis*. — Livid eruption, countenance almost black, tongue coated dark brown, sordes on the teeth, inability to protrude tongue (J. F. MILLER).

*Mercurius* is indicated where gastro-intestinal symptoms predominate. The tongue is heavily coated, showing the imprints of the teeth; breath very offensive; diarrhœa of slimy stools, with tenesmus. Also bronchitis, with loose, barking cough and no expectoration; offensive sweats; diphtheritic angina.

*Pulsatilla* may be indicated early, although its sphere of usefulness lies mostly in the clearing up of the cough and catarrhal symptoms lingering after measles. It is followed well by *Hepar*.

*Veratrum viride*.—"During febrile stage, especially if pulmonary congestion is impending; red streak down centre of tongue; convulsions before eruption" (C. G. R.).

Other remedies which may be called for upon special indications are:

*Belladonna*.—May be indicated early, but less frequently than *Aconite* in mild cases. Nervous symptoms predominate, and convulsions occur at the eruptive stage.

*Carbo veg.*—Persistent hoarseness remaining after measles.

*Drosera*.—Cough occurring in paroxysms in the afternoon, spasmodic and attended with bloody or purulent expectoration.

*Hepar* and *Spongia* may be required when the cough becomes croupy.

*Phosphorus* and *Antimon. tart*, in those cases in which broncho-pneumonia predominates.

*Sulphur*.—Either during the first stage, when the eruption is tardy, or for the sequelæ, such as chronic coughs, originating in the remnants of partial pneumonia; chronic diarrhœa; hardness of hearing and chronic ear discharges (C. G. R.).

#### SCARLET FEVER.

Scarlet fever is a highly contagious, infectious disease of childhood, characterized by fever, angina and a diffuse scarlet



eruption, followed by desquamation. It is endemic in all large cities, often breaking out in epidemics. The greatest degree of susceptibility exists between the ages of two and six; infants usually escape, especially those nursing at the breast, while in children nearing puberty the susceptibility gradually decreases. One attack gives immunity to a second, as a rule. Epidemics are most prevalent during the fall and winter months.

While scarlet fever is not as infectious as measles, its spread being slower and less extensive than that of measles in communities or non-isolated quarters harboring cases, still its *contagiosum vivum* possesses much greater tenacity to life, and is much more readily carried from one location to another by means of a third person or by contaminated objects. It retains its vitality for months, and requires active germicidal measures for the successful disinfection of infected localities and articles of dress, bedding, etc.

The period of contagiousness lasts about six weeks, beginning with the invasion of the disease, reaching its height during the febrile period and persisting until desquamation is complete. The source of infection lies in the catarrhal discharges, the scales of epidermis, and probably also in the excreta. The contagion may persist in the expectoration or nasal secretion even after the stage of desquamation.

The exact nature of the causative agent of scarlet fever still remains obscure. Streptococci are found in the blood in a certain percentage of cases, but they are rather to be looked upon in the light of a secondary infection than as the primary cause of the disease. Hektoen (*Jour. Amer. Med. Ass.*, March, 1904) isolated streptococci from twelve out of a hundred cases. They occur with relatively greater frequency in the more severe and protracted cases, but they may be absent in some of the fatal cases. Mallory (*Jour. of Med. Research*, Jan., 1904) claims to have demonstrated certain bodies in the skin of four cases of scarlet fever, which he looks upon to be one of the stages in the development of a protozoon. The period

of *incubation* is short, usually less than a week, and in many cases only one to two days.

**Symptomatology.**—The course of a typical case of scarlet fever may be divided into the *stage of invasion*, *stage of eruption* and *stage of desquamation*. Prodromata are rare, the invasion being abrupt, with repeated chills, followed by high fever, headache, prostration and vomiting, together with sore throat. Such a combination of symptoms occurring in a child should always lead one to suspect scarlet fever. The temperature may rise very rapidly to a high point, reaching 104° F. and over; in mild cases, however, it may rise but inconsiderably. The pulse likewise is affected in a characteristic manner, attaining a rapidity of one hundred and twenty to one hundred and forty beats per minute quite early in the attack. The throat is highly inflamed, a diffuse erythematous blush covering the tonsils, pharynx and soft palate. Later on, diphtheritic patches are liable to appear.

Within from twelve to thirty-six hours from the beginning of the fever the *eruption* makes its appearance, first showing about the neck and chest, whence it rapidly spreads over the entire body, this being accomplished within twenty-four to thirty-six hours, or in even a shorter period of time. The eruption appears most intense on the neck, over the extensor muscles, about the joints, and on the dorsum of the hands and feet. A peculiar pallor about the mouth is frequently seen, producing a striking contrast with the flushed cheeks, and giving rise to the characteristic "white line" of the disease. The eruption is due to intense hyperæmia of the skin, accompanied by exudation of round cells into the rete Malpighii and serous exudation, the process ending in death of the epidermis, with desquamation of variously-sized scales and flakes. The predominating feature in the pathology of the cutaneous manifestations is vascular paralysis. When typical, the rash consists of numerous, closely-aggregated red points, the size of a pin-head, evenly distributed over the entire body, giving it a bright, scarlet color. The eruptive points may be but

slightly red in the beginning, later assuming the bright, scarlet hue. The rash is more frequently a dull red than scarlet, and the general effect is produced by the erythema associated with puncta, fine vesicles and more or less goose-flesh. The punctate spots are the result of inflammation around the hair follicles, and they may become large enough to impart to the skin a distinctly rough feel. The points may be flat or elevated, round or lentil-shaped, and with increasing hyperæmia they become confluent, the skin becoming turgescent and tense. The swelling is most marked about the face and eyes in these cases (*scarlatina lævigata*). This is the variety for which Hahnemann recommended *Belladonna* as both prophylactic and curative, while for *scarlatina miliaris*, a variety in which there are minute papules interspersed with fine vesicles filled with a turbid serum, he recommended *Aconite* (HARTMANN, *Kinderkrankheiten*), considering it a special variety of scarlet fever. Another deviation from the usual eruption is the appearance of roseola-spots of various sizes and shapes, separated by pale areas of skin (*scarlatina variegata*). In some cases the rash does not become general, often being absent from the face in mild cases. It may be extremely faint in color, or assume a deep purplish hue, or become hæmorrhagic.

At the height of the eruption the skin is burning hot to the touch, and the patient complains of burning, stinging and itching; at this time, also, all other symptoms are most intense.

Pressure with the finger causes momentary disappearance of the rash, which reappears from the periphery toward the center, differing in this respect from the rash of measles. In cases marked by prostration the peripheral circulation is so poor that the rash only slowly reappears after having been obliterated by pressure. This is a valuable prognostic sign.

The *temperature* curve of scarlet fever is one of abrupt onset, the fever running high with very little remission during the first three or four days and then gradually subsiding by

lysis so that at the end of a week the temperature is again normal.

The *tongue* is thickly coated white; the edges, however, remaining red. In the course of a few days the coating is shed, leaving the red and swollen papillæ exposed, with the resulting characteristic appearance described as "strawberry-tongue." Enlargement of the papillæ of the tongue is such a constant symptom of scarlet fever that it becomes a most valuable diagnostic sign. Indeed, McCollom, of Boston, looks upon this symptom when occurring in association with fever and sore throat as pathognomonic of scarlet fever, irrespective of the presence of a rash. In mild cases, however, the enlargement of the papillæ may fail to develop.

Should the *throat* become seriously affected at this time, patches of membrane will be seen upon the tonsils which may spread to the soft palate and adjacent parts. This complication is usually due to streptococci, true diphtheria being rare during the course of scarlet fever, and, when associated with the same, occurring as a sequela rather than as a complication.

*Otitis* is a frequent complication occurring at the height of the disease, the result of an extension of infection from the angina. It usually terminates in suppuration, and is one of the commonest causes of deafness in children. When occurring during convalescence its advent is more readily anticipated, as there is recurrence of fever, with distinct earache and impairment of hearing.

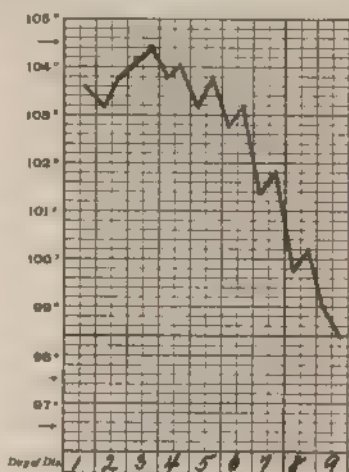


FIG. 58. — TEMPERATURE CHART FROM A CASE OF SCARLET FEVER

*Parotitis* and *cellulitis of the neck* sometimes accompany the septic process in the throat. The termination of such a process is usually in suppuration. Likewise the tonsils and lymphatic glands of the neck may share in the suppurating process, rendering the prognosis most unfavorable.

*Synovitis* of the larger joints is prevalent during some epidemics. It develops between the first and second weeks. The duration is short, never ending in suppuration. Beside this condition, an attack of *acute articular rheumatism* is frequently invited in individuals of the rheumatic diathesis, occurring as a complication of the scarlet fever either during the eruptive stage or during convalescence.

The *blood* shows a well-marked leucocytosis, the polynuclears predominating. The more intense the infection the higher the leucocytosis. In asthenic cases, however, there may be a failure on the part of the organism to react and in such cases a low leucocyte count offers a grave prognosis. In such cases the eosinophiles may be decreased or absent (DA COSTA).

The *lymphatic glands*, both the subcutaneous as well as the lymphatic structures of the viscera are involved. There is more or less general adenopathy, the cervical, inguinal and axillary glands being especially affected.

*Post-scarlatinal nephritis* is one of the most constant and most important complications of scarlet fever, occurring typically during the third week. Pathologically, it is an acute, diffuse, productive nephritis. It is a more serious condition than the simple acute degeneration or acute exudative nephritis which may occur early in the course of the fever, just as in any other acute infectious disease. There is scanty urine and general dropsy, and suppression of urine and acute *uræmia* may supervene. Although the kidney is much damaged at the time, still a marvellous degree of regeneration may set in and the child shows a fair chance of "growing out" of the disease, so to speak, under careful treatment.

*Desquamation* begins shortly after the rash has faded—about

the end of the first week. It begins in the localities in which the rash first appeared, showing itself as scales of varying size about the neck and chest. Gradually the entire trunk is involved in the process, desquamation being completed here long before the fingers and toes have shed their dead epidermis. In these parts, especially where the skin is thick, the peeling process is slow, and large pieces of skin, sometimes complete casts of the fingers, are detached in the "moulting" process. In cases where desquamation is slight, it may be found characteristically by about the tenth day at the tips of the fingers. A separation of the epidermis at the edge of the nail-bed, producing the line of "subungual cleavage," is a characteristic phenomenon.

The *prognosis* depends to a great extent upon the character of the epidemic; the general health of the child before the attack; the height of the fever, and the severity of the attending complications. As a rule, the disease is more liable to prove fatal if the child is very young, especially when serious throat implication, nasal diphtheria, diarrhoea or otitis are associated. The degree of toxæmia and the state of the peripheral circulation are important prognostic indications. A livid, sluggish rash or recession of the rash, indicating failing circulation, are unfavorable signs. Cases marked by sudden onset with excessively high fever offer a grave prognosis on account of the high degree of toxæmia they present. Some cases prove fatal within the first twenty-four hours before the rash appears—"malignant scarlet-fever."

Among the later dangers are especially to be feared nephritis, which displays a tendency to develop particularly in cases in which cutaneous manifestations are mild, probably because the scarlatinal toxins are more actively excreted through the kidneys than through the skin in these cases. Should uræmic convulsions supervene, either death or cerebral hæmorrhage with resulting hemiplegia, etc., may result. Otitis always brings with it danger of cerebral abscess. The patient is also liable to develop true diphtheria at this time.



Convalescence is usually protracted owing to anæmia, chronic otorrhœa and nasal catarrh, hypertrophied tonsils, post-scarlatinal nephritis.

**Diagnosis.**—Scarlet fever differs from *measles* in the abruptness of its onset, the absence of Koplik's sign and prominent catarrhal symptoms, and the characteristic appearance and behavior of the eruption alluded to in the symptomatology of both affections. The scaling in scarlatina is also different from that observed in measles. From *rubella* it is distinguished by the sudden onset and high fever with pronounced sore throat, by the characteristic appearance of the tongue, and by the occurrence of desquamation. *Symptomatic rashes* can usually be traced to the partaking of certain articles of food or the administration of certain medicines, or to septic- or auto-intoxication. The rash is of short duration, sore throat is absent, and in the absence of gastric derangement the temperature is normal. Many of the infectious fevers are at times accompanied by an erythematous rash, causing considerable confusion as to the true nature of the case. All doubtful cases, however, followed by the typical desquamation and associated with albuminuria, are to be looked upon as scarlatina.

The history of exposure to infection is an important datum in atypical and incomplete cases, as is also the appearance of the tongue and the presence of general adenopathy. The presence alone of scaling is not a proof that the case is one of scarlet fever, and scaling may be more pronounced in certain cases of desquamative scarlatiniform erythema than in ordinary scarlet fever. The time of onset, mode of progress and its persistence are of more importance than the mere presence of scaling (SCHAMBERG). On the other hand, in a case of scarlet fever with well-developed rash and subsequent marked desquamation, the associated conditions, namely, fever, prostration, sore throat and adenopathy, are more pronounced than in the *scarlatiniform erythemata*.

**Treatment.**—With the occurrence of suspicious symptoms



the patient should be isolated immediately. From this time on until desquamation is completed, and, if practicable, until all catarrhal discharges have been controlled, the child should be kept away from others to whom or through whom it may convey the contagion. Six weeks from the beginning of the attack is usually a sufficiently long period of quarantine; but, just as with the classical ten days of the lying-in period, there is liability to variation in either direction.

The bedroom should be freely ventilated, and all unnecessary articles of furniture and hangings should be removed, but not after they have been exposed to the contagion, unless they can be immediately disinfected. A sheet wrung out of a 2 per cent. solution of Carbolic acid and hung in front of the door adds to the completeness of the isolation. All kitchen utensils, etc., used by the patient should be immersed in a 4 per cent. solution of Carbolic acid or Formaldehyde for an hour before being removed from the room. They should then be scalded, or, still better, boiled for a quarter of an hour. The nurse and the attending physician should protect their outer clothing by donning a long, linen coat on entering the sick room, and disinfect their hands before leaving the room. All sheets, rags, articles of clothing and furniture that can be dispensed with are best burned. For disinfection of the room after its vacation by the patient there is nothing equal to Formaldehyde gas generated in the Schering lamp from pastilles. If Sulphur be used, one pound must be burned for each hundred cubic feet of room space; at the same time steam should be generated, the room of course being hermetically sealed during the operation. It is always wise to precede the fumigation by a thorough mopping of the floors with a 1 to 2,000 bichloride solution, allowing it to dry in situ. If the walls are papered, they should be scraped down and repapered.

During the occupation of the room by the patient the spraying of hydrogen dioxid with an atomizer greatly aids in keeping the air pure. If the patient suffers much from angina or

laryngitis it will prove advantageous to generate steam, at the same time placing dishes of slaked lime about the room.

“The terrible burning and itching of the skin is best relieved by rubbing the body all over with bacon, olive oil or cocoa-butter, once or twice a day; always if the skin is dry, glands swollen, and there is a scrofulous diathesis.” (C. G. R.) I would object to the use of carbolized oil or other powerful antiseptic applications to the skin at this time, its action being necessarily injurious and its efficacy in destroying contagion questionable. The inunction of fats not only relieves the itching and burning of the skin, but it also acts as a sedative and at times reduces the fever.

In case of high fever a sponge-bath of tepid water and alcohol (one part of alcohol to three of water) is of great service. In the advent of anasarca or suppression of urine a warm pack should be used. (See Treatment of *Acute Nephritis*, p. 371.) For the angina, a spray of alcohol one part, glycerin one part and water four parts, may be used several times daily. Likewise, the nose should be kept scrupulously clean by means of douches of a normal saline solution or Dobell's solution. These simple measures may prevent ulceration and suppuration in the throat, and also suppurating otitis media. Pseudo-diphtheria developing, it should be treated with *Permanganate of Potash*, as recommended under *Diphtheria*.

“As a preventive I would still recommend the potentized *Belladonna*, one dose every night, until symptoms appear. If it cannot prevent the attack, it has seemed at least to mitigate its violence.” (C. G. R.)

The *diet* should be restricted to a non-nitrogenous one as far as possible, in order to relieve the kidney of any extra strain in its excretory work. Solid food, especially meat, should be prohibited until after the third week, and in case of nephritis developing, a milk diet must be adhered to for a still longer period.

The remedies of first importance in scarlet fever are the following:

*Aconite*.—*Aconite* was recommended by Hahnemann in scarlatina miliaris. High fever; great restlessness and anxiety; whining and moaning; delirium, with irrational talking; anorexia; mouth and throat dry; pharynx and tonsils deep-red color; skin hot and dry. The eruption in these cases does not correspond to the diffuse, smooth redness characteristic of *Belladonna*, and, with full development of constitutional symptoms, the condition usually goes over into a typical *Rhus* state. Personally I do not believe that the character of the rash is of much importance in prescribing and I pay more attention to the other manifestations, namely, the degree of fever, prostration, nervous irritability, angina, etc. For this reason *Belladonna* is a much better remedy in the early stages of the vast majority of cases than *Aconite*.

*Arsenicum*.—Eruption tardy, scanty, or becoming petechial. Adynamic cases, with putrid sore throat; nephritis; dropsy; typhoid state. The usual characteristics of the remedy are present.

*Belladonna*.—" *Belladonna* is only indicated in the smooth form of eruption with vascular and nervous excitement; it does no good in adynamic cases. The miliary form of eruption is much more adapted to *Amm. carb.*, *Lach.* or *Rhus tox.*" (C. G. R.) There is congestion of the brain, with active delirium; sudden starting in sleep; bright, glistening eyes; throbbing of the carotids; cerebral congestion; tongue coated white, with red edges, the papillæ showing through the coating; bright redness of throat, with swelling and dysphagia; pungent heat of skin, with moisture on covered parts. Indicated in the majority of cases in the beginning of the disease and if the case be a mild one, no change of remedy will be required. Otherwise it is usually followed by *Rhus tox.* (pronounced toxæmia); *Apis* (anasarca); *Mercurius iod. rubr.* (pseudo diphtheria), etc.

*Bryonia*.—Delayed appearance of eruption; face crimson red; mouth and lips dry; tongue dry and brown; great thirst; the child wishes to lie perfectly quiet and undis-

turbed. *Bryonia* is frequently indicated when rheumatism, synovitis or involvement of the pleura and meninges complicate the case.

*Carbolic acid* is highly recommended by Goodno.

*Cuprum*.—Sudden recession of the eruption, with occurrence of cerebral symptoms. The *Acetate of Copper* is generally preferred. The *Arseniate of Copper* should always be thought of when the condition is one of uræmia.

*Gelsemium*.—In the early stages, when there is the characteristic dullness and drowsiness; aching and prostration; soft, compressible pulse; aching in the eyes and back of head. The throat is red and feels swollen; the eyes are suffused, and the patient feels chilly, especially along the spine.

*Lachesis*.—Scarlatina miliaris. Eruption becoming purple and livid; desquamation delayed; hæmaturia (*Terebinthina*); oppression when lying down; diphtheritic complication; diarrhœa, with foul-smelling stools.

*Rhus tox.* may be indicated from the beginning when the rash is not of the smooth, diffuse variety, and, instead of vascular and nervous excitement, there is prostration, with great restlessness; high temperature, with drowsiness; tongue red and smooth; epistaxis; œdematous swelling of the skin in various parts, the eruption becoming dusky with the development of miliary vesicles; swelling of the cervical glands and cellular tissue about the neck; ulceration of the throat.

*Sulphur*.—Intense redness of entire body, like a boiled lobster; skin hot and dry, with great burning.

*Veratrum vir.*—In the beginning, when there is great vascular excitement, wiry pulse, dilated pupils, convulsions. The pulse is hard and wiry, arterial tension being greater than in *Aconite*, while anxiety and restlessness are less marked.

*Zincum* is indicated where the eruption is scanty, of a pale bluish-red color or entirely absent, while cerebral symptoms are pronounced. "Especially in the anæmic; brain exhausted; not able to develop exanthemata" (HERING). Meningitis in

the stage of paralysis. Convulsions followed by stupor; the feet are in constant motion, or the child lies perfectly motionless, with eyes open, pupils dilated, cornea insensitive. When these symptoms are present there is little to be hoped from any remedy; but if we can anticipate them, and give *Zincum* on its early indications, a fatal termination may be averted.

Remedies less frequently indicated, but of great importance in special cases, are:

*Ailanthus*.—Miliary rash; small, rapid pulse; the eruption becomes dark and livid; intense angina, with acrid discharge; muttering delirium followed by stupor.

*Arum triph.*—Tongue red and swollen, acrid discharge from nose; diphtheria, swelling of submaxillary glands; the corners of the mouth and the lips are cracked, and the child picks at the lips and finger-nails until they bleed.

*Ammon. carb.*, *Apis*, *Lycop.*, *Muriatic acid*, *Opium*, *Phos.*, *Phos. ac.*, *Phytolacca* and *Stramonium* also bear a strong relationship to special symptoms.

**Complications and Sequelæ.**—*Throat complications* call for *Phytolacca*, the various salts of *Mercury*, *Kali bichrom.*, *Permanganate of Potash*, *Lachesis* and others. (See *Diphtheria*.)

*Cellulitis* and *Parotitis*.—The most important remedy for this complication is *Rhus tox.* Suppuration calls for *Hepar*, *Mercurius*, *Silica*.

*Otitis*.—*Bell.*, *Puls.*, *Rhus tox.*, *Pantago*. Cerebral complications, *Apis*, *Bell.*, *Helleb.*, *Hyos.*, *Stram.*, *Sulph.* and *Zinc*.

*Enterocolitis*.—*Mercurius* usually controls the diarrhœa, but *China*, *Rhus tox.*, *Veratr. alb.* may also be indicated.

*Nephritis*.—*Cantharis* is a most valuable remedy in post-scarlatinal nephritis when there is not much blood in the urine and only moderate dropsy. When the latter is pronounced *Apis* and *Arsenicum* are of greater service. The characteristic "smoky" appearance of the urine frequently seen after scarlet fever, from the free admixture of blood, is a strong indication for *Terebinthina*. Persistent albuminuria after scarlet fever calls for *Mercurius corr.*

## RUBELLA.

Rubella, *Rötheln*, or *German Measles*, is characterized by moderate fever, sore throat, and an exanthem which in some instances resembles that of measles (*rubella morbilliforme*), and in others that of scarlet fever (*rubella scarlatiniforme*). Complications or sequelæ are scarcely ever observed. It usually occurs epidemically, and one attack gives immunity against another, but in no wise protects against measles or scarlet fever.

Nothing definite is known of its *etiology*. It is contagious, but less so than measles or scarlet fever; nevertheless it may be spread by articles of clothing, etc. Infants under six months are immune. The incubation period averages two weeks, but it may show considerable variation in this respect.

**Symptomatology.**—The period of invasion is short, prodromata usually being absent. Drowsiness, slight fever and sore throat precede the eruption by a day or more in some cases; in others the rash appears before the child has shown evidence of any illness. It is first seen upon the face, from which it spreads over the entire body in the course of twenty-four hours. Although the face is the most constant site of the eruption, even when the rash is developed but partially, still the chest and back may show the first signs of eruption in exceptional cases. The duration is about three days. Often it has completely faded from the face by the time the lower extremities are involved.

In *rubella morbilliforme* there is seen a discrete, maculopapular rash of pale red color, the eruptive points being slightly elevated and about the size of a pin's head or larger. These lesions have a tendency to become confluent upon the face, particularly so when they are numerous.

In *rubella scarlatiniforme* the rash is of a diffuse, uniform, scarlet color, never as intense, however, as in scarlet fever, and with unmistakable evidence of the maculopapular eruptive points in various localities (on the forehead, fingers and toes, and about the wrists).



Desquamation occurs to a slight degree after deflorescence of the rash, but in mild cases it may be entirely wanting.

Catarrhal symptoms are not a necessary accompaniment of rubella, and throat symptoms may be so slight as to remain unnoticed. The slight cough present is due to an infection of the mucous membrane and tonsils, as in la grippe (KOPLIK). In a number of my cases there was decided follicular pharyngitis, and in some a slight exudate was present upon the mucous membrane. The superficial lymphatic glands of the posterior cervical and posterior auricular region are transiently swollen, this being one of the characteristic symptoms of the disease. Usually there is also involvement of the axillary and inguinal glands.

The duration is short, seldom over five days. The *prognosis* is good; complications are rather to be considered accidental than otherwise.

In many instances the *diagnosis* can only be made after the mild course of the disease has been noted, in conjunction with the absence of complications and sequelæ, especially if an epidemic is not known to be on at the time. When, however, we are aware of such an epidemic, and especially if the child has previously had one of the other exanthemata, the diagnosis presents little or no difficulty. From *measles* it is chiefly to be differentiated by the absence of catarrhal symptoms, absence of Koplik's spots and the slight fever. From *scarlatina* the absence of the strawberry-tongue, the rash first appearing upon the exposed portions of the body, the low temperature and absence of desquamation and nephritis readily differentiate it.

The *treatment* is simple in a frank case of rubella, but until we are aware of the true nature of the case the child should be cared for identically as in a case of measles or scarlet fever, in order to be on the safe side. Cases resembling measles will require remedies suited to mild cases of the same (*Aconite*, *Bryonia* or *Pulsatilla*), and those resembling scarlatina will usually require nothing more than a few doses of *Belladonna*.



## VARIOLA; VARIOLOID.

Variola, or *small-pox*, is an acute infectious, highly contagious disease, characterized by fever of a typical course, vomiting, intense lumbar pains, and an eruption of papules passing through the stages of vesicles, pustules and crust formation, the vesicles being umbilicated.

The nature of the *contagion* has not been determined. It is contained in the secretions, excretions and exhalations of the body, being especially disseminated by means of the dried scales and contents of the pustule. Pfeifer and others have constantly found small, homogeneous bodies in the epithelial cells surrounding the lesions. One or two are usually found in the cell substance. They probably belong to the class of protozoa (PARK).

It attacks all ages, from the foetus *in utero* to the aged. A case came under my notice in which the eruption appeared in a new-born infant on the fifth day. During the last three weeks of her pregnancy, the mother had had an attack of varioloid, which was overlooked at the time on account of its mild nature. The infant died on the twelfth day. Among children it proves especially fatal. One attack protects against another, at least for a long period of time. The period of incubation is from nine days to two weeks.

The pock first consists of an area of round-cell infiltration into the *rete mucosum*, in which a central area of coagulation-necrosis takes place. Inflammatory reaction occurs around this area, which represents the central depression of the vesicle, with the formation of a reticulated vesicle containing serum, leucocytes and fibrin filaments. Pustule-formation supervenes, the leucocytes and cells of the rete mucosum becoming necrotic.

**Symptomatology.**—The invasion is marked by a severe chill or repeated chills, in children, often convulsions, with rapidly rising temperature. In children, convulsions are common at this period. Vomiting and intense backache

are accompanying symptoms. "In some epidemics the initial stage is marked by an erythematous eruption, either diffuse or measly, or by a hæmorrhagic exanthem which consists of extremely small punctate, often pin-head sized hæmorrhages into the epidermis, at times so closely crowded together that the impression of a diffuse redness is produced." The temperature rises on the first day to  $103^{\circ}$  to  $104^{\circ}$  F., continuing with slight morning remissions until the evening of the third day, when it reaches its highest point. On the fourth day it falls several degrees, this remission lasting until the seventh or eighth day, when there is a secondary rise—the suppurative fever.

The stage of eruption commences on the evening of the third day. "There appear little red spots first in the face. If very numerous they coalesce, like measles-spots, with which they might be confounded if it were not for the granulated feel which they present to the sense of touch (like shot)." (C. G. R.)

The eruption rapidly spreads to other portions of the body, and on the third day of eruption the papule is converted into a clear vesicle presenting an umbilication at its summit. The vesicle is also loculated. In the course of a few days (eighth day of the disease) the vesicle is transformed into a pustule, which dries up after a few days or breaks down, with the formation of a soft, yellow crust, later becoming brownish and dropping off, leaving a somewhat elevated spot which in time entirely disappears. This occurs where the lesions are discrete and where the process has not extended into the deeper layers of the skin. Here they adhere for a long time, leaving an uneven scar, which at first looks pink, but by degrees grows conspicuously white, to remain so throughout life.

Simultaneously with the appearance of the eruption upon the skin, identical lesions develop upon the mucous membranes exposed to the external air. Here it may result in great destruction of tissue.

Small-pox may run its course as a discrete, confluent, hæmorrhagic, gangrenous or malignant variety. The modified variety occurring in those partially protected by vaccination, and running a mild course without secondary fever, is described as *varioloid*. In every other respect it is identical with true small-pox.

The *prognosis*, excepting in *varioloid*, is always grave. As complications may be mentioned broncho-pneumonia, pleurisy, septicæmia, ulcerating keratitis, suppurating otitis, arthritis.

The *diagnosis* is often rendered difficult by the primary erythematous eruption. The true eruption may be confounded with *measles* in its early stages, but the sensation of balls of shot under the skin imparted to the finger by the papules of small-pox is a pathognomonic distinction, beside the severe initial symptoms of the attack. Again, in *measles* the temperature rises to its acme with the appearance of the rash, while in small-pox there is a temporary drop in the fever as the rash comes out.

From *varicella* it is distinguished by the intensity of its symptoms. Moreover, the eruption appears later than in *varicella*, does not come out in crops, is distinctly umbilicated, and presents a well defined inflammatory areola. The eruption of small-pox is also decidedly harder and more palpable than that of *varicella*.

**Treatment.**—As small-pox is one of the most serious and most dreaded of all contagious diseases, every precaution to prevent a spread of the same must at once be instituted when we are confronted by a suspicious case. The most rigid isolation and disinfection, as described under *Scarlet Fever*, must be carried out to the letter. Besides this, every person in the house not recently successfully vaccinated (within four years) should immediately undergo the operation. The patient must have as much fresh air as possible. If the fever is very high sponge-baths are indicated. Osler (*Practice of Medicine*) has come to the conclusion that the

prevention of pitting is really not within the hands of the physician. Protecting the ripening papules from light and keeping the hands and face covered with lint soaked in cold water or mild antiseptic lotions is, however, to be recommended. The *red-light* treatment exerts no influence over pustulation (SCHAMBERG). Later on, we should aim to prevent the crusts from becoming hard and dry by the free application of vaseline. The addition of a little Carbolic acid or Boric acid to the vaseline is a distinct advantage.

In the early stages, *Aconite*, *Bell.*, *Bry.*, *Gelsemium* and *Rhus tox.* are to be recommended. Jahr (*Therapeutische Leitfaden*) began all cases with *Variolinum* as soon as the diagnosis could be established; and if, in spite of this remedy, the course became a grave one, he followed with *Sulphur*. He preferred these two remedies above all others.

*Vaccininum* is spoken of favorably by Goodno and others.

From a limited personal experience with small-pox I have come to look upon *Bryonia* followed by *Rhus tox.* as the treatment most likely to exert a favorable influence over the disease. In the stage of suppuration when toxæmia sets in *Cinchona* tincture and whisky should be freely used. When collapse threatens it may become necessary to resort to *Strychnia*.

#### VACCINIA.

Vaccinia, or *Cow-pox*, is an eruptive disease of the cow, inoculable into man, and producing a lesion at the site of the inoculation resembling the pock of variola, together with constitutional disturbances. No specific germ for vaccinia is known, nor is the true nature of the disease understood, some considering it a primary disease of the cow, while others believe it to be small-pox modified by its passage through animals. It has been experimentally demonstrated that children vaccinated with cow-pox were not susceptible to inoculation with small-pox virus, the reverse condition also holding true. Pfeiffer and others have found small homogeneous bodies in

the epithelial cells surrounding the lesions of both small-pox and vaccinia, and as small-pox virus has produced in cattle a disease indistinguishable from cow-pox, there is hardly any doubt that the two are due to the same micro-organism, modified by its transmission through the cow (PARK).

A successful inoculation with vaccinia affords protection against small-pox in the majority of cases, at least for a number of years. Small-pox occurring in those who have been vaccinated usually assumes a mild course, *i. e.*, varioloid. As to the modifying influence of vaccinia upon small-pox already in progress there is a difference of opinion. According to Marson, if a person exposed to small-pox be vaccinated within four days, small-pox will be prevented; if later, but early enough to allow the vesicles to reach the stage of areola, the attack of small-pox will be modified; but later than this it is useless. Curschmann opposes this view as erroneous. It is interesting to know the views expressed by Hahnemann on this subject, which are no doubt borne out by the most trustworthy clinical testimony—"It is well known that when variola is added to cow-pox, the former, by virtue of its superior intensity as well as its great similitude, will at once extinguish the latter homœopathically and arrest its development. Cow-pox, on the other hand, having nearly attained its period of perfection, will, by its similitude, lessen to a great degree the virulence and danger of a subsequent eruption of small-pox, for which we have the testimony of Mühry and many others" (*Organon*).

The operation of vaccination consists of the introduction of the lymph from the vaccine vesicle of heifers into the circulation by bringing it in contact with a scarified surface for a sufficient length of time to permit of its absorption. Having cleansed the site of inoculation (the usual seat is the left arm, just below the insertion of the deltoid muscle) with soap and water, followed by scrubbing with alcohol or ether, a few parallel scratches about half an inch in length are made with a sterilized needle, just deep enough to break the

epidermis and expose the *rete mucosum*. A drop of *glycerinated vaccine lymph*, this being the most reliable and aseptic form in which the virus can be obtained, is placed upon the scarified surface and rubbed in gently with the needle. Guest (*Pediatrics*, Vol. IX, No. 5) has arrived at the conclusion that the entire contents of a tube is too large a quantity of lymph for the average child, judging from the results obtained in four hundred cases vaccinated by this method, in which there was more pronounced inflammatory reaction and more glandular swelling, besides the formation of a larger scab than in his former cases inoculated with points. I have, however, found that the old-fashioned ivory point usually causes a more severe lesion than the lymph, although in order to avoid an aggravated form of vaccination we must not scarify too freely or rub in too much lymph. After permitting the seat of inoculation to dry, the scarification is covered with a piece of sterilized gauze, over which a shield or bandage is applied. By the adoption of this careful method, complications and sequelæ rarely, if ever, follow.

**Symptomatology.**—During the first three days after the operation, nothing excepting a slight local irritation, soon subsiding, will be noticed. On the third day, however, a papule appears at the site of inoculation, surrounded by an areola; this papule is converted into an umbilicated vesicle on the fifth or sixth day. The vesicle attains its maximum development by the eighth day, after which it becomes pustular. The areola gradually increases in size and depth of color until this time, but disappears as the acute symptoms subside. The pustule then dries up, forming a scab. On the twenty-first day the scab comes off, leaving the characteristic deep, circular, pitted scar.

The constitutional symptoms accompanying vaccinia are fever, malaise, anorexia, etc., which begin with the eruption, and attain their height at the period of pustulation, after which they rapidly disappear. Swelling of the axillary glands is usually present.

Variations from the above-described course frequently occur. The vesicle may be late in developing, may be premature and not fully developed; a generalized pustular eruption may accompany the primary lesion, which may persist in recurring attacks after healing of the same; or complications, notably erysipelas, ulceration and sloughing, glandular abscesses and septicæmia, may develop as the result of faulty technique. Vaccinia may also occur as a general eruption of papules, which turn into vesicles and pustules. They appear on the face and extremities about the fifth day. I have also encountered a general papular rash occurring on the tenth day, looking like measles or the early stage of small-pox.

Deaths have occurred, but they were almost invariably from avoidable causes, as Voigt shows in his statistics. There is always a risk, however, in vaccinating a delicate, sickly child, and the operation should never be performed when an acute disturbance is present, or if there is a case of contagious disease in the family to which the child has been exposed. I have observed some anti-vaccinationists vaccinate, and their careless method has convinced me that they had good cause to be dissatisfied with this practice.

Besides, the invaccination of syphilis (when humanized virus was used) has occurred, and claims have been made that tuberculosis was likewise transmitted. This, however, has not been proved, although vaccination may have been, in some instances, the exciting cause in stirring up a latent tuberculous lesion in strumous and tuberculous children into an acute condition.

The age at which children are vaccinated is usually the third month, in the absence of any acute or constitutional illness. In the absence of an epidemic of small-pox I do not see the necessity for so prompt a procedure. It is quite early enough to vaccinate the child after it is out of its teething difficulties, and some physicians, believing in the efficacy of vaccination to control whooping-cough, keep it in reserve to be employed as the opportunity manifests itself. The child



should, however, be vaccinated before it is sent to kindergarten or school, and revaccinated at the period of puberty, or on the occurrence of an epidemic of small-pox.

**Treatment.**—After vaccination I give *Aconite*, following the same with *Belladonna* if fever, headache, diffuse redness and swelling about the site of eruption and glandular swelling develop. *Apis* or *Rhus* may be indicated by erysipelatous manifestations. After the acute symptoms have subsided it is well to give a few doses of *Sulphur*, or if the scab separates with suppuration and an unhealed ulcer remains, *Silica*. I firmly believe that when vaccination is carried out on strictly aseptic lines, and the child is watched throughout as in the case of any other illness—being put to bed if necessary, and carefully prescribed for—none of the many complications and so-called constitutional after-effects, attributed to vaccination, will follow. The complications and sequelæ of improper vaccination and the constitutional disturbances caused by the same will require symptomatic treatment. The remedies most frequently recommended are *Thuja*, *Silica*, *Malandrinum* and *Sulphur*.

#### VARICELLA.

Varicella, or *chicken-pox*, is an acute infectious disease characterized by the eruption of discrete vesicles, which appear in crops, and disappear, in the course of a few days, by desiccation.

The specific virus has not been isolated, but it is known to exist in the vesicles, and can be transmitted by inoculation. The usual manner of contracting the disease is through contact with a case, although a third person may carry the infection. One attack protects against another. It may occur sporadically or epidemically. The period of incubation is usually two weeks.

The *symptoms* are slight in the majority of cases, but they may assume such a grave nature in delicate children, especially in the tuberculous, that the diagnosis may present some

difficulty. However, the subsequent course of the disease will remove all confusion in the matter. The onset is abrupt, as a rule, the first signs of the disease being the appearance of papules and vesicles upon the trunk and extremities, accompanied by slight fever, anorexia, coated tongue and languor. Constitutional symptoms may be so slight as to attract no attention. Each day a new crop of vesicles makes its appearance; this usually continues for three or four days.

The eruption appears first as a small red papule, soon becoming vesicular. The vesicles are unilocular, although at times multilocular vesicles are seen. They are surrounded by a faint areola, and do not become pustular unless infected by scratching, etc. In the course of a few days they dry up, the crusts soon falling off without leaving a scar, although in some cases a circular, pale area is left, which persists for some time, or, if ulceration has taken place, quite a conspicuous scar may remain.

*Varicella gangrænosa* is a type of varicella which is attended by gangrenous stomatitis, as a result of infection in poorly-nourished or tuberculous children. If the process becomes extensive, it may prove fatal. As complications—which, however, are fortunately rare—may be mentioned erysipelas, adenitis, cellulitis, gangrenous dermatitis and nephritis. It is not uncommon to have varicella and one of the other infectious fevers occur simultaneously, although the error must not be made of considering those cases of varicella beginning with an erythematous or measles-like rash as cases of varicella plus scarlet fever or measles.

**Diagnosis.**—Varicella is to be differentiated from *small-pox* by the slight constitutional disturbances accompanying the rash, which appears abruptly, coming out in crops, and soon disappearing by dessication, without pustulation or scar-formation. The eruption of small-pox does not always come out at once, and frequently new papules and vesicles will continue to appear for several days after the first lesions were seen. They do not, however, erupt in distinct crops, nor do

we find lesions in the various stages of development, that is, fresh papules and vesicles interspersed among pustules, as is to be observed in varicella. Again, the papules of varicella lack the shot-like feel characteristic of the small-pox lesion, and the vesicles are more delicate and present a characteristic pearl-like appearance. If the vesicle has not dried up by the fourth day, it is more likely small-pox than varicella.

**Treatment.**—In the presence of fever, rest in bed, a light diet, and, when there is much itching, the use of rye-flour as a dusting-powder, or olive oil and Boric acid, is about all that is required in mild cases. *Aconite* may be called for in the beginning, to be followed by *Rhus tox.* The gangrenous or pustular variety will call for *Arsenicum*, *Mercurius*, *Rhus tox.*, etc.

#### PERTUSSIS.

Pertussis, or *whooping-cough*, is an acute infectious disease in which there is present a catarrhal process of the respiratory tract and a characteristic paroxysmal cough. It occurs both epidemically and sporadically, infection taking place through close proximity to a case; seldom through the agency of a third person. Close proximity, however, is necessary, as the air does not seem to convey the contagion to any great distance about the patient. Epidemics are said to occur every eighteen months or two years in large cities, as in measles.

Several micro-organisms have been credited with being the exciting cause. Afanassjeff isolated a short bacillus, the *bacillus tussis convulsivæ*, but failed to demonstrate satisfactorily the reproduction of whooping-cough by inoculations with pure cultures of this bacillus. The investigations of Czaplewski point to another bacterium as the *contagium vivum*; this bacterium is two or three times as long as broad, rounded and somewhat thickened at its ends, is divided in the middle, and surrounded by a capsule in its natural state. The secretions of the normal mucous membrane of the nose contain very few bacteria, while in whooping-cough we find

a large mass of this particular kind, in fact, a natural pure culture (WAGNER, *N. Y. Med. Jour.*, Oct. 8, 1898). The most recent studies of the secretion expelled after a coughing paroxysm have shown a short, ovoid bacillus, similar in appearance to, and of the same group as, the influenza bacillus. The bacillus grows best upon blood-agar and agglutinates with the blood of pertussis patients in as high as 1-200 dilution (MARTHA WOLLSTEIN).

The *contagion* exists mainly in the sputum, and the patient should be considered capable of spreading infection as long as the cough retains its characteristic paroxysmal nature. The period of incubation is from one to two weeks.

The *pathological processes* accompanying whooping-cough are catarrhal inflammation of the larynx, particularly in the region of the interarytænoid cartilages; tracheitis and more or less bronchitis; swelling of the bronchial glands; rhinitis. In fatal cases broncho-pneumonia with emphysema and areas of atelectasis are the most common lesions found; there may also be entero-colitis and cerebral congestion, with effusion and cortical hæmorrhages. The toxin of whooping-cough in some cases appears to affect the smaller blood-vessels and favor hæmorrhagic extravasations, either spontaneous or as a result of the congestion which is associated with the cough-paroxysm. Moebius believes that the nervous system may also be acted upon by this toxin in a manner somewhat similar to the action of the diphtheria toxin.

**Symptomatology.**—The course of whooping-cough is in three stages: the premonitory, or catarrhal; the spasmodic, and the stage of decline. The first stage usually lasts ten days to two weeks; the second stage may persist for a month or more; while the stage of decline is a gradual transition into an ordinary bronchial cough, which varies with the state of the child's health and with the season of the year. The average duration of an ordinary case is, therefore, about six weeks, but the course is greatly influenced by treatment and also by the advent of complications.

The attack begins as an ordinary cold, indistinguishable in the beginning from a simple bronchitis, with, however, this difference, that instead of yielding to treatment in the course of a few days, or abating of its own accord, the cough gradually increases in frequency and severity, soon assuming the paroxysmal and spasmodic type characteristic of the disease. An early symptom that should always arouse suspicion is the nocturnal aggravation of the cough from the very beginning.

Examination of the chest at this time reveals nothing beyond a slight bronchitis. In the very beginning there is usually indisposition, running of the nose, a short, dry cough, and slight fever. These symptoms soon abate, but the cough increases in severity. The cough is characterized by a sudden, loud expulsive effort, followed in rapid succession by similar efforts of gradually decreasing force; through these continued explosions the chest is almost completely emptied of air, so that the child is obliged to draw in a deep breath at the end of the paroxysm. As the glottis is narrowed during this long-drawn inspiration, a loud, piping sound is produced, constituting the whoop, from which the disease is named. As soon as the lungs have been refilled the cough begins anew, consisting, as before, of rapidly following expulsive efforts, ending with the whoop. This continues (two to six coughing fits) until the paroxysm is terminated either by the dislodgement of a plug of mucus from the trachea, or by the vomiting of the ingesta or of a quantity of tenacious mucus.

During such an attack the face becomes red, even livid; the eyes are injected and bulging, and the child clings to the nearest object for support, or stands with the feet wide apart and the hands resting upon the knees. Bleeding from the nose frequently occurs during the paroxysm, and cortical hæmorrhages from the meningeal vessels are to be feared in violent cases. When such a hæmorrhage is extensive, hemiplegia and convulsions will follow. This hæmorrhagic ten-

dency is one of the most serious aspects of whooping-cough. Sub-conjunctival hæmorrhage is quite common. No doubt the action of the pertussis toxin upon the blood-vessels is responsible for the condition.

The number of paroxysms in a day will vary from only a few to as many as fifty. They are usually more frequent during the night. In very young children the cough is not as characteristic as in older ones, the whoop being especially faint or indistinct, but the same paroxysmal nature of the cough is present, and, indeed, they may suffocate during a severe spell.

There are signs upon which we can base a fairly positive opinion as to the existence of whooping-cough in most cases, even without having heard the cough. But it is rarely necessary to exclude this pathognomonic symptom, for should the child evince no desire to cough during our examination it is but necessary to press the finger into the jugular fossa, or irritate the pharynx with a tongue depressor, in order to bring on a paroxysm. The face appears bloated from the recurring vascular enlargement, and the eyes are deeply injected; slight hæmorrhages may be seen under the conjunctiva. The eyes are unnaturally moist. Under the tongue a characteristic sign is frequently seen, namely, ulceration of the frænum. This is induced by the repeated propulsion of the tongue over the lower incisor teeth in coughing. In my experience it has only been present when there was at the same time catarrhal stomatitis in association with the whooping-cough, rendering the mucous membrane particularly vulnerable.

With the decline of the disease the paroxysms become less frequent and less severe, soon losing the spasmodic character of the cough, and the expectoration becomes mucopurulent, as in an ordinary bronchitis. With a fresh cold the whoop may reappear; this, however, is to be considered rather as an intensification of the cough in a subject in whom the spasmodic habit has been formed than as a true recurrence of the disease.

The commonest complications of whooping-cough are *broncho-pneumonia* (in the winter months) and *entero-colitis* (summer months). The advent of broncho-pneumonia is recognized by the appearance of fever, together with rapid respirations and dyspnoea, and subcrepitant râles throughout the chest. The cough may change during the height of such a complication, assuming more the incessant, dry or rattling character belonging to broncho-pneumonia. In a case seen in consultation a five year old child was suddenly seized during the fourth week of whooping-cough, with a high fever and cerebral symptoms so pronounced as to suggest meningitis. Examination of the chest revealed *croupous pneumonia* of the right upper lobe.

*Diarrhoea* is liable to become a troublesome symptom in delicate children, often leading to marasmus.

*Convulsions* due to extreme general nervous irritability are frequent among infants. They may, however, be due to asphyxia, meningeal hæmorrhage (see p. 511), or pneumonia, giving the case an entirely different aspect. *Meningitis* rarely, if ever, results from whooping-cough, although marked meningeal symptoms due to hyperæmia of the brain and œdema of the pia mater may be observed.

*Dilatation of the heart*, due both to the strain on the heart as well as to the action of the toxin upon the myocardium, may be observed (KOPLIK).

As a *sequela*, tuberculosis is most to be dreaded. Whooping-cough, as is well known, is one of the most potent predisposing causes of tubercle, ranking second to measles in this respect. This is due to the fact that in both of these diseases inflammation of the bronchial glands occurs prominently.

The *prognosis* depends to a great extent upon the age and previous health of the child. Normal children above five years of age seldom suffer great inconvenience or serious after-complaints under proper treatment. The prognosis becomes grave when broncho-pneumonia is added, or where the hæm-



orrhagic tendency is marked; and in infants (notably the rachitic and tuberculous) the prognosis should be guarded.

**Diagnosis.**—During the prevalence of an epidemic the diagnosis should present no difficulties. Isolated cases, however, may become puzzling, especially when atypical. The character of the cough, together with the accompanying signs described under the symptomatology, should bear one out in differentiating whooping-cough from an ordinary bronchitis. The pertinacity and intensity of the cough, with the absence of all other signs indicating a thoracic condition commensurate with such a cough, is characteristic.

Prof. Filatow, of Moscow, confirms the researches of Hippus and Blumenthal, who noticed that pertussis patients have a *pale urine* of *high* specific gravity.

*Hyperplasia* of the *bronchial glands* frequently provokes a paroxysmal cough, but the course is a chronic one, and there is associated bronchitis, and usually tuberculous foci elsewhere in the chest. Other possibilities of error are found in the so-called "*spasmodic bronchitis*" of infants, and *catarrhal laryngitis* (false croup).

**Treatment.**—Isolation is difficult to carry out, as the disease is already contagious during the stage at which it cannot always be recognized. Nevertheless, every effort should be made to protect delicate children and infants against exposure by excluding from their presence, during an epidemic, all children with suspicious colds or hacking coughs.

The patient should receive as much air as possible, and in pleasant weather may be permitted to be out-of-doors. Protracted cases do well from a change of climate, the seashore being particularly beneficial.

If the cough is very troublesome at night, and especially in the case of infants in whom asphyxia is to be feared, the vaporizing of *Cresoline*, *Creasote* or *Oil of Eucalyptus* in the sick-room is often attended with the happiest results. Holt prefers Creasote vaporized in a croup-kettle; a weak Formaldehyde vapor is also of service at times in mitigating the paroxysms.

The *remedies* recommended for whooping-cough are legion, and space forbids enumeration of so long a list. While there are, perhaps, a dozen which are used a hundred times when the others are used but once, still it is impossible to tell just which remedy will be of the greatest benefit in a given case before the symptoms have been carefully considered. The popular feeling as to the clinical value of our remedies in this affection is well presented by the following statistical report by Dr. Geo. B. Peck (*Trans. American Institute of Homœopathy*, 1898): "Out of every thousand prescriptions by members of this Society for the amelioration of that group of morbid phenomena popularly designated whooping-cough, at least 175 are for *Drosera*, 153 for *Belladonna*, 123 for *Ipecacuanha*, 76 for *Cuprum* (*metallicum* and *aceticum*), 54 for *Corallium rubrum*, 44 for *Antimon. et pot. tartaricum*, 24 for *Mephitis*, 20 each for *Aconitum napellus* and for *Hyoscyamus*, 18 for *Naphthalin*, 15 for *Coccus cacti*, 13 for *Kali bichromicum*, 11 for *Bryonia*, 9 for *Magnesia phosphorica*, 8 for *Chelidonium majus*," etc.

In the early stages *Aconite*, *Bell.*, *Ipecac.* or *Tartar emet.* may be indicated. As soon as the true nature of the case becomes apparent a remedy should be given capable of controlling the course of the disease. Opinions differ as to the most potent remedy to accomplish this result. No doubt the remedy will vary with the epidemic, and while *Drosera*, *Belladonna*, *Naphthalin*, etc., are useful in many instances, they are not invariably so. As soon as the cough is accompanied by the raising of secretion I am in the habit of prescribing *Tartar emetic.*, 2x trit., unless indications point strongly to another remedy, such as *Ipecac.*

If, in spite of the administration of one of the above-mentioned remedies, the case continues steadily to advance or become of a more serious type, *Cuprum*, *Mephitis*, *Corallium rubrum*, *Coccus cacti* and *Hyoscyamus* should be thought of. Protracted cases will often yield to *Carbo veg.* with remarkable promptness.

*Ambra grisea*.—Hollow, paroxysmal cough, with expectoration of tough, grayish or yellowish mucus, especially after awaking in the morning ; belching after cough.

*Anacard*.—Ill-natured children, with uncontrollable temper ; cough brought on by fits of vexation (*Ant. crud*.—Great irritability ; disagreeable toward those of whom it was formerly very fond, even striking at them.)

*Arnica*.—Painful paroxysms (*Bryonia*) ; tendency to hæmorrhages ; meningeal hæmorrhage.

*Bell*.—Intense redness of face during paroxysm ; nervous erethism ; convulsions ; eyes bloodshot ; cough deep and hollow ; sneezing after cough. The most important remedy in the early stage.

*Carbo veg*.—Protracted cases. Follows well after *Drosera*. Hoarseness ; anæmia ; sluggish circulation ; flatulent indigestion.

*Coccus cacti*.—Cough, especially worse in the early morning, followed by the expectoration of yellowish or bloody, tough mucus (*Ambra grisea*). I have had excellent results with this remedy during the paroxysmal stage, when there was abundant stringy, yellowish expectoration.

*Cuprum*.—Convulsions ; the paroxysms are severe and long-continued, the child becoming blue in the face ; cerebral complications (follows well after *Ipecac*).

*Drosera*.—Paroxysmal stage. Worse after midnight ; gagging and vomiting predominate ; the expectoration is frequently blood-streaked ; tuberculous diathesis. Personally, I have been disappointed in the results seen from this remedy.

*Hyos*.—Incessant cough when lying down, relieved by sitting up.

*Ipecac*.—Spasm of the glottis before paroxysm ; the child stiffens out during the cough and becomes blue in the face (a strong indication for *Ipecac* in my experience). Bronchopneumonia, with abundant fine râles ; vomiting after cough. The expectoration is often blood-streaked. Hughes recommends beginning all cases with *Aconite* and *Ipecac* in alternation.

*Mephitis*.—During the spell the child passes both urine and fæces; diarrhœa and flatus very offensive; the child must be taken up during the cough, turns blue in the face and seems asphyxiated. In a number of grave cases in infants, in whom suffocation seemed imminent, *Mephitis* in the second decimal dilution has given me excellent results.

*Naphthalin*.—Goodno recommends this remedy to be used as soon as the case is recognized. He employs the first decimal trituration.

*Tartar emetic*.—Broncho-pneumonia. Rattling of mucus in larger tubes; gasping for air; deficient oxygenation of blood. I have in late years obtained the best results from this remedy as a routine prescription when another remedy was not strongly indicated.

*Sulphur* may be required in the third stage, if the patient relapses into his former condition on the slightest provocation (*Carbo veg.*).

*Phenacetine* and *Antipyrine* are much used by the old school. Hale recommends *Phenacetine* in the ix trituration, two to ten grains every three to four hours.

#### PAROTITIS.

Epidemic parotitis, or *mumps*, is an acute infectious disease in which the parotid glands are attacked by an intense catarrhal inflammation. The specific contagion is not known, but it no doubt gains access into the gland through the duct of Steno, setting up an intense hyperæmia, followed by a profuse serous exudation (soft swelling). The process begins in the ducts and acini of the gland, rarely extending to the interstitial connective tissue, and only terminating in suppuration when there is an accidental infection with pyogenic microorganisms accompanying the primary infection. For this reason resolution is perfect in the vast majority of cases, as the tumefaction is the result simply of hyperæmia and œdema and not of structural changes in the gland.

**Secondary parotitis** is an infection of the parotid gland (usually one-sided), with pyogenic micro-organisms, occurring during the course of one of the infectious fevers. It may complicate typhoid fever, diphtheria, scarlet fever, small-pox and measles, rendering the prognosis most grave. In these cases the submaxillary gland is rarely spared. Unlike as in mumps, it terminates in suppuration, the entire parenchyma of the gland being more or less involved in the destructive process.

Mumps appears epidemically, although never to the extent attained by epidemics of the other prominent contagious diseases of childhood. Close contact seems necessary for infection. It is most prevalent during the damp seasons and among those living in damp dwellings. The period of incubation is from two to three weeks. One attack gives immunity against another.

**Symptomatology.**—For a day or two there may be a slight fever with lassitude, restless sleep, nervous irritability, loss of appetite, etc., preceding the appearance of the characteristic lesion. The inflammation of the gland induces first a painful stiffness of the jaw and tenderness in the region of the parotid. Swelling rapidly sets in, and in the course of a few days the gland will be swollen to its utmost extent. The fever may increase and the sleep become disturbed by restless dreams or delirium; convulsions have been known to occur in young children. The left parotid is the one most frequently attacked first. In the majority of cases the opposite side begins to swell in a day or two after the appearance of the first lesion. Sometimes the opposite parotid is not involved until the first begins to subside, or it may escape entirely.

At the height of the disease the face presents a ludicrous appearance. The entire parotid region stands out prominently from the presence of a tense, shining swelling which spreads anteriorly to the zygoma and posteriorly to the sternocleido-mastoid. The tumor feels firm over its centre while

the edges pit on pressure. The enlargement is uniform and regular, not nodular as in lymphadenitis. It is also perfectly immovable, for the parotid gland is so firmly held down by the deep fascia as to render its displacement impossible.

The fever now gradually subsides, usually not lasting more than from three to four days, but the patient is extremely uncomfortable, every effort at opening the mouth being attended with pain, and any article of food not bland in character frequently exciting intense suffering. In fact, the excruciating pain produced by taking anything acid into the mouth is looked upon as pathognomonic and a symptom of diagnostic value. The swelling attains its height within three or four days, subsiding by the end of a week. This, as has been above stated, is accomplished rapidly and perfectly, and persisting permanent structural changes should lead to a suspicion of a mixed infection (*staphylococci* or *tubercle bacilli*).

Metastases to the testicle in the male and to the ovary or breast in the female are not uncommon in older children at this time, *i. e.*, during the stage of decline, but in young children this does not occur. Aside from the possibility of such a complication the *prognosis* is good.

**Secondary parotitis** occurs during the course of one of the acute infectious diseases, and begins as a hard, painful swelling, more circumscribed than in mumps, with an inflammatory blush soon showing itself over the surface. This gradually deepens in color; the swelling becomes more tense, and points of fluctuation can be elicited. In the cases which I have seen the sub-maxillary gland was also involved. One case, complicating typhoid fever, proved fatal. If allowed to open spontaneously there is a free discharge of thin, sanious pus. The *prognosis* is always grave, although it is said to be less so when occurring later in the course of the disease which it complicates.

**Diagnosis.**—It seems unnecessary to call attention to the question of diagnosis in a simple case of mumps, yet errors

are sometimes made. One of the most frequent is the mistaking of acutely enlarged *cervical lymphatic* glands for mumps; here the slower onset, the multilocular feel of the tumefaction and its movability will readily distinguish this condition from mumps. *Diphtheria*, with pronounced swelling of the cellular tissue of the neck, has likewise been mistaken for mumps, as I have personally witnessed. The possibility of such an error occurring can only impress us most forcibly with the importance of a routine inspection of the throat in every acute febrile disease of childhood.

**Treatment.**—The most important remedy is, no doubt, *Belladonna*. It corresponds to the vascular engorgement, the fever, and the nervous irritability so common in mumps.

*Mercurius* may be indicated early when there is but slight fever, pale swelling of the parotid region and gastric derangement. It is useful in the later stages of all cases to hasten resorption of the exudate.

For metastasis to the testicles *Pulsatilla* and *Clematis* are the chief remedies. If induration with tendency to atrophy follows, *Aurum* should be considered.

Metastasis to the ovaries calls for *Apis*, *Colocynthis*, *Pulsatilla*, *Hamamelis*.

*Secondary parotitis* finds in *Rhus tox.* its most appropriate remedy. As the process advances, *Hepar* or *Arsenic* usually becomes indicated. *Calc. sulph.* is the main remedy to promote healing after pus has been discharged either through fistulous openings or by means of an incision. As soon as the gland becomes swollen, hot fomentations wrung out of a 1 to 4,000 solution of the Bichloride of Mercury should be applied continuously. This offers a hope of aborting, or, at least, limiting the process.

#### INFLUENZA.

Influenza, or *la grippe*, is an infectious disease occurring pandemically and attacking all ages alike. It is characterized by fever of sudden onset and short duration, accompanied



by marked prostration and complicated with either catarrhal inflammation of the respiratory or alimentary tract, or by certain nervous phenomena. This is the true influenza, and it is to be distinguished from those endemic cases of so-called grippe, catarrhal fever or epidemic bronchitis which occur in children with great regularity every year, especially during the fall and winter months.

The *bacillus of Pfeiffer* is the exciting cause, being found in almost pure culture in the sputum of freshly infected cases. It is a short, thin rod with rounded ends; it does not stain by Gram's method and is best demonstrated with dilute fuchsin. It is difficult to cultivate; besides, it usually disappears from the sputum early and for this reason its presence is often missed.

The period of *incubation* is short, seldom exceeding a few days. One attack does not afford immunity against another, as is the case in many of the epidemic infectious diseases; on the contrary, it may even lead to an increased susceptibility to a fresh attack, or, at least, to acute catarrhal affections.

While influenza, as a rule, pursues a short and acute course, nevertheless it shows a tendency to become protracted in many instances, sometimes becoming latent for a while and then suddenly flaring up with acute manifestations. Again, bronchitis may persist for weeks, the secretion showing influenza bacilli in pure culture (ORTNER, *Modern Clinical Medicine*, 1905) and pneumonia of a protracted course may likewise be due to the influenza bacillus, these cases presenting particular difficulty in their differentiation from pulmonary tuberculosis (WASSERMANN). Again, certain cases of protracted catarrh of the respiratory tract running their course under the type of remitting and intermitting fever were first recognized by Filatow as a chronic form of influenzal infection (*Vorlesungen u. Infectious-Krankh. im Kindesalter*, 1897).

**Symptomatology.**—The disease begins abruptly with fever, severe headache, general aching and prostration. The fever remains at its height for a period of from three to five days,

in the absence of complications, during the entire course of which prostration is marked, and headache and muscular aching are usually very distressing. A symptom present at this time and upon which Fürbringer, of Berlin, lays great stress, is marked redness of the face. This shows itself as a diffuse flush and differs from scarlet fever in the absence of the white line about the mouth and pallor of the forehead. As Fürbringer also points out, there is often present a slight icteric discoloration of the skin, although there is not much evidence of bile in the urine. I have in a few instances observed actual jaundice develop during influenza. Several clinical types are to be encountered, depending upon the predominance of catarrhal or nervous symptoms and the locality chiefly attacked.

Thus, there is the *cerebral form*, characterized by a predominance of headache, together with delirium, and even unconsciousness, some of these cases simulating meningitis; the *abdominal form*, characterized by vomiting, anorexia, gastralgia, diarrhœa, some with predominance of gastric symptoms, others simulating typhoid fever; the *neuralgic form*, in which there are neuralgic pains in the peripheral nerves and other regions; the *thoracic form*, complicated by broncho-pneumonia, and the *catarrhal form*, the commonest variety, in which catarrh of the upper respiratory tract is the most prominent symptom. Extreme prostration, however, is common to all forms, this being the chief feature of the disease. The toxin exerts a most potent influence upon the nervous system, which manifests itself as prostration, cardiac weakness and neuralgic pains, and during convalescence in the persisting prostration and the strong tendency to the development of neurasthenia, perineuritis, insomnia, persistent headache, and even insanity. Fortunately these complications are not as common in children as in adults, and, taken altogether, the prognosis is better, although a complicating broncho-pneumonia may change the entire aspect of the case. As in the case of measles and whooping-cough, a predisposition to infection with the tubercle bacillus is created.

*Nephritis* may occur in influenza; sometimes this is of the hæmorrhagic type.

*Rhinitis* and *otitis* (see p. 532) are frequent troublesome complications.

The *pneumonia* complicating influenza is a most serious affection, as a rule leading to diffuse and catarrhal inflammation of the finest tubes with pronounced dyspnoea and toxæmia, while consolidation is inconsiderable. Lobar pneumonia, however, appears to occur with striking frequency during grippe epidemics and the two diseases may occur simultaneously (see p. 284, 290). Pleurisy, with "clay-water effusion", (FÜRBRINGER), and abscess of the lung may complicate such a pneumonia.

The prognosis depends upon the age of the patient, the previous health and the presence of complications. Filatow lays stress upon the fact that in childhood it is mainly during the first to third year that the grave cases are encountered.

The diagnosis seldom presents difficulties during the prevalence of an epidemic, but isolated cases may be mistaken for a variety of other affections, particularly in the beginning. The catarrhal symptoms, hard cough and drowsiness may lead to a suspicion of beginning *measles*, but the subsequent course soon corrects this error. From *pneumonia* it is to be distinguished by the absence of physical signs indicating lung involvement, absence of extreme prostration and comparatively short course. The majority of cases simulate pneumonia more closely than any other disease, and a careful, daily physical examination of the chest is necessary in order to differentiate the two affections. Bacteriological examination of the sputum and nasal secretion may or may not throw a positive light upon the subject. Cerebral cases may simulate *meningitis* or *cerebro-spinal meningitis*. The mild cases of *grippe* above alluded to present none of the profound toxic manifestations of influenza, being nothing more than an infectious rhinitis or bronchitis. In *protracted cases* the condition is often very puzzling. Such cases especially simulate tubercu-

losis. Here the absence of physical signs of tuberculosis and the bacteriological examination of the mucous secretions are the most conclusive diagnostic data.

In cases of influenza in which pulmonary consolidation occurs as a complication the differentiation from primary *croupous pneumonia* rests upon the following data, according to Filatow (*loco cit.*): (a) Mode of onset, whether with catarrhal symptoms or with a chill. (b) The presence of an epidemic or occurrence of other cases of influenza in the same house. (c) The time of occurrence of the physical signs of consolidation, *i. e.*, whether demonstrable in the first three to five days or not until a later period. (d) The age; in children under three years croupous pneumonia more frequently complicates influenza than in older children, in whom it is more likely to be primary. (e) The clinical course; influenzal-pneumonia runs a protracted, irregular course; the fever is remitting and relapses are common.

**Treatment.**—The child should be put to bed immediately, absolute rest enforced, and great care taken to avoid exposure to cold or draughts, in order to ward off serious pulmonary complications.

The diet should be highly nutritious, but to prevent gastrointestinal complications, easily digested food only should be selected. When the pulse becomes weak and irregular a moderate amount of whisky should be administered at regular intervals.

During convalescence much can be done to ward off the many sequelæ belonging to influenza by rebuilding the child's constitution as quickly as possible with appropriate diet, remedies and hygienic measures. During convalescence, and in protracted cases, there is nothing more desirable than a change of climate, the seashore being particularly beneficial to these patients.

The most important remedies for the average case are *Aconite*, *Gelsemium* and *Bryonia* in the beginning (*Gelsemium* and *Bry.* frequently suffice for the entire course of the

disease), and later we must choose from such an array as *Arsenicum*, *Euphrasia*, *Allium cepa*, *Phosphorus*, *Kali bichrom.*, *Rhus tox.*, *Antimon. tart.*, *Pulsatilla*, etc., according to the preponderance of disturbances of a certain type or in certain localities.

*Arsenicum* is indicated where the prostration is extreme and presents the chief manifestation of the disease. This is often seen in infants. There may also be sneezing; acrid, watery coryza; the process extending to the chest, with cough and dyspnœa; great restlessness.

*Belladonna*.—Cerebral cases; starting in sleep, delirium, throbbing headache.

*Bryonia*.—Pains in the muscles, every limb aching intensely; lies perfectly quiet and does not wish to be disturbed; dry, painful cough. Broncho-pneumonia complicating influenza (*Ant. tart.*, *Phosphorus*).

*Eupatorium perf.*—Deep-seated aching in the back and extremities, as if the bones would break; the skin is slightly jaundiced and the tongue heavily coated; bilious vomiting.

*Gelsemium*.—The symptomatology of *Gelsemium* presents a true picture of the average case of grippe. The condition begins with lassitude and chilliness; "creeps" especially up and down the back, and the patient hugs the stove to get warm. He feels prostrated, every part of the body aches, and he complains of headache, soreness and sensitiveness of the eyes, obstruction of the nose, sore throat and prostration. The full, low tension, rapid pulse, heavy eyelids and flushed appearance of the face are very characteristic of *Gelsemium*. *Baptisia* should be administered if this condition does not promptly improve under *Gelsemium*.

*Pulsatilla*.—Catarrhal symptoms predominate; mild, tearful disposition; the tongue is heavily coated and covered with viscid saliva, but there is no thirst; the patient is constantly chilly; diarrhœa.

*Rhus tox.*—Aching in the limbs, causing great bodily restlessness; cannot remain quiet in one position; prostration and typhoid symptoms.

*Sanguinaria* is often indicated when the rhinitis and pharyngitis are intense. There is fluent coryza, involvement of the accessory nasal cavities (sinusitis), dryness of the throat with burning, extending down into the œsophagus; wheezing cough.

During convalescence *China* is a valuable remedy. It aids in restoring the patient's former strength and appetite. Giserius (ARNDT, *Practice of Medicine*) obtained most satisfactory results from its use, in the first decimal dilution, in several Berlin epidemics.

#### EPIDEMIC CEREBRO-SPINAL FEVER (MENINGITIS); SPOTTED FEVER.

As the name implies, this is an inflammatory affection involving the meninges of the brain and spinal cord and occurs epidemically, although sporadic cases are not uncommon, especially in localities that have once been the seat of an epidemic. The disease does not appear to be contagious and during epidemics the cases occur in irregular distribution, there being no evidence that they are communicated from one individual to another. The exciting cause is the *diplococcus intracellularis meningitidis* (meningococcus) of Weichselbaum. It must not be forgotten, however, that there are other forms of purulent meningitis which often bear a striking clinical resemblance to this specific disease. In this connection, *pneumococcus meningitis*, which may be primary, deserves special mention, as it has no doubt often been mistaken for cerebro-spinal fever.

As to the virulence of the meningococcus, this is very slight, as Heubner has shown. This explains why this form of meningitis is the least fatal, and why epidemics never attain the numerical strength reached by other infectious diseases when they break out epidemically.

The *diplococcus meningitidis* was discovered in 1888 by Weichselbaum and its rôle as the specific micro-organism in the disease is now universally accepted. Morphologically it

resembles the gonococcus of Neisser, being biscuit-shaped and found within the polynuclear leucocytes. It readily stains with Loeffler's methylene blue and decolorizes by Gram's method, although not constantly (JAEGER). It is cultivated with difficulty, growing best on Loeffler's blood serum at body temperature. Weichselbaum is of the belief that the diplococcus gains access to the brain by way of the nose, ear and upper air-passages. This seems plausible, as the organisms have been found in the nasal secretion of victims of the disease; also, rhinitis may be one of the early symptoms of cerebro-spinal fever.

The bacteriologic diagnosis is of the greatest prognostic importance. Cases of meningitis in which the pneumococcus is present usually die very rapidly, while those due to the diplococcus intracellularis run a more protracted course and often recover. The high mortality of some epidemics is probably due to the inclusion of many cases caused by Fraenkel's organism (LENHARTZ). The nasal secretion may contain the organism, but the only reliable method of procedure is to examine the fluid obtained by lumbar puncture (see p. 458).

Epidemics of cerebro-spinal meningitis occur most frequently in the winter months. While country districts and barracks have been mainly attacked in the past, still our large cities are now getting their share of the disease. Children are peculiarly susceptible to the disease and it may attack young infants. Poverty, overcrowding, physical overexertion and even traumatism have been looked upon as predisposing causes.

**Pathology.**—Rapidly fatal cases show only slight pathological lesions. On the other hand, fully developed cases show all the signs of a severe, purulent meningitis. Some cases become protracted and result in thickening of the meninges; degenerative changes in the cerebral cortex; marked distention of the ventricles.

The pathological process is an exudative inflammation of



the pia mater affecting chiefly the base of the brain and the posterior surface of the cord. The exudation into the cord is most pronounced in the dorsal and lumbar region. Effusion into the ventricles and into the pia mater of the cortex co-exists to a lesser degree. The cranial nerves are more or less involved according to the amount of exudate and pressure entering into the case. The exudate is at first sero-fibrinous, soon becoming purulent. It is rich in polynuclear leucocytes. Some degree of cerebritis may co-exist.

Associated lesions that may be encountered are cutaneous hæmorrhages (petechiæ); nephritis; broncho-pneumonia; parenchymatous degeneration of the heart, liver and kidneys; arthritis.

**Symptomatology.**—The disease is most irregular in its clinical manifestations and may prove fatal within a few hours or run a long and protracted course. There are intermediate cases of moderate severity in which perfect recovery takes place. Epidemic cerebro-spinal meningitis is the least fatal form of meningitis, but unfortunately a larger proportion of the cases that recover are left with some permanent disability such as deafness, blindness, idiocy, paralysis.

A number of types of cerebro-spinal meningitis are to be recognized, the classification being based chiefly upon the duration and severity of the symptoms. The invasion, however, shows a general resemblance in all cases. The onset is sudden, with either vomiting or convulsions, intense headache and fever, and soon the most characteristic symptoms of the disease, namely, rigidity of the neck muscles and retraction of the head, makes its appearance.

In the *fulminating* form the onset is so sudden and overwhelming that the patient may succumb within a few hours.

These foudroyant cases usually prove fatal in the first few days. Deep coma develops early and is associated with retraction of the head and even opisthotonos. Strumpell (*Specielle Path. u. Therapie*) mentions a class of cases with sudden and grave onset, similar to the fulminating form,

which however abort in the course of several days and go on to complete recovery (*abortive form*). Then there are *mild cases*, in which the entire clinical course is marked by slight development of the symptoms, the duration also being shorter than usual.

*Protracted cases* are not uncommon. The symptoms may extend over a period of from two to three months and the child ultimately recover. In these protracted cases the fever often disappears entirely for days and the other manifestations will abate, only to recur with renewed vigor. When the fever becomes decidedly intermitting, they are spoken of as *intermitting cer. bro-spinal fever* (VON ZIEMSEN). The last quoted course is possible, because of the remissions that take place in the pathological process, which may be compared with the repeated relapses observed in cases of rheumatism pursuing a chronic course (HEUBNER, *Kinderheilkunde*). The same author reports the case of a boy who after three and a half months of suffering made a complete recovery. *Emaciation*, attaining a marked degree of *marasmus*, goes hand in hand with the progress of the disease.

The *ordinary form* runs an average course of from two to four weeks. It is so irregular, however, that it is impossible to foretell the outcome of any case. While the onset is as a rule sudden, still there may be some prodromal manifestations—such as malaise, and headache; slight fever. In infants, conjunctivitis is not infrequently observed.

The initial symptoms point to the brain as the seat of the affection. They are vomiting or convulsions, sometimes a chill; intense occipital headache and high fever. To these symptoms stiffness of the neck is soon added and with the outpouring of exudate more or less disturbance of consciousness is added.

In infants bulging of the fontanel is to be noted. *Delirium* is a common symptom with older children. Complete stupor develops, although often the patient may be aroused, or lucid moments will alternate with the stupor. Fever and delirium

may only be present at night, the child being rational during the day and able to sit up in bed and play with its toys (KOPLIK).

The headache may be so intense that we will observe the child knitting its brows and moaning with pain, while at the same time it is so deeply in stupor that we can neither arouse it nor get a response. Beside the cerebral manifestations we will observe marked *hyperæsthesia* of the cutaneous surface, due to the irritation of the posterior nerve roots (spinal) by the inflammatory exudate. This hyperæsthesia is most marked in the lower extremities.

Retraction of the head occurs earlier (within a day or two or even within a few hours) and is more pronounced and more persisting in cerebro-spinal fever than in any other form of meningitis (HEUBNER). As a rule, there is tenderness along the entire spine, which may be rigid or arched.

Disturbances in the functions of the cranial nerves are manifested as hyperæsthesia of the sensory and irritation of the motor nerves. Thus photophobia, tinnitus aurium and disturbances of smell are encountered. Optic neuritis may develop with consequent blindness, and permanent deafness is another of the unfortunate sequelæ of the disease.

Spastic strabismus; irregular but reacting pupils; ptosis; spasm of the facial muscles and dysphagia are all to be observed. In contradistinction to tuberculous meningitis, there is more tendency to irritation and less to actual paralysis in cerebro-spinal meningitis than in the former.

The extremities are rigid, the arms usually being flexed while the legs are straightened and resist passive movements. If, however, we flex the thigh upon the abdomen, or if the patient attempts to get up, spasmodic flexion of the leg upon the thigh takes place. This phenomenon is known as *Kernig's sign*. It is readily demonstrated by flexing the thigh upon the abdomen with the patient in the dorsal position, and then attempting to extend the leg out on a line with the thigh. The method of obtaining Kernig's sign in cerebro-spinal meningitis is shown in Fig. 46; ordinarily, however,

the thigh should be flexed to a greater degree than here shown, in order to avoid all possibility of error. As the leg is lifted up in this manner a spasmodic resistance is encountered, due to contracture of the hamstring muscles. Kernig's sign is present in a large proportion of all cases of meningitis, but especially when the meninges of the cord are at the same time involved. It indicates irritation of the pyramidal tracts, Fraenkel (New York) thinks the phenomenon depends upon traction on the cauda equinæ, and he calls attention to the fact that we often can see the Babinski sign take place in the foot simultaneously with the occurrence of the Kernig.

The *cutaneous* manifestations are important, and we should not forget that the disease acquired its old name from the petechial rash that is present in about a third of the cases. In Osler's cases the rash was common. Fully one-half, if not more, present herpes labialis or facialis (STRUMPELL).

The *fever* does not conform to any regularity of type. It usually rises rapidly with the onset of the disease, reaching to 102° to 103° F., even in the fulminating cases, but the temperature does not bear a constant relationship to the severity of the case. As Goodno says, "In none of the infectious fevers is the average temperature as low as it is in cerebro-spinal fever, and in none are such remarkable fluctuations manifested." Cases have been observed in which practically no fever was present. The diurnal variations may be of wide range and the highest point is not necessarily attained in the evening hours, as is usually the case in other infectious diseases. The intermitting type resembles pyæmia rather than malaria. In the protracted cases the fever may abate for several days and then recur with all its former vigor. In Osler's experience a sudden fall of temperature is a bad omen.

The *pulse* may be slow in the beginning, but the characteristic slowing observed in tuberculous meningitis is not encountered. In fulminating cases, however, it may be slow

and irregular. This is a grave symptom. Usually it is rapid and irregular. The *respirations* show nothing pathognomonic; exceptionally Cheyne-Stokes respiration occurs.

The *blood* shows a distinct leucocytosis.

**Complications** are observed in some cases, they are more common in some epidemics than in others. Aside from the complications on the part of the nervous system already referred to there may be clonic contractions and paralysis of individual extremities and occasionally unilateral paralysis. They appear to be due to various pressure conditions by meningeal exudates or possibly they owe their origin to variations in circulatory conditions, but at the autopsy a clear insight as to what has caused these paralyses is by no means always obtained (EICHHORST, *Modern Clinical Medicine*).

*Pneumonia* and *arthritis* may occur as metastatic inflammations. The arthritis of cerebro-spinal meningitis closely resembles acute articular rheumatism and may be associated with endocarditis.

As *sequelæ*, long-continuing nervous disturbances, such as vertigo; headache; loss of memory; neurasthenia are common. Permanent deafness, blindness, idiocy and chronic hydrocephalus have been referred to.

**Prognosis.**—Excepting in the mild and abortive cases the prognosis is most grave. Even if the acute symptoms subside there is the danger of the case becoming protracted and running into a fatal *marasmus* or of one of the unfortunate sequelæ remaining after recovery. There are few recoveries in children under two years (KOPLIK). In older children the mortality is about 40 per cent. (HIRSCH).

**Diagnosis.**—In fulminating cases that die before the clinical picture of the disease is developed, it is naturally impossible to make a diagnosis. When during an epidemic, however, a child is seized with fever, vomiting or convulsions and rapidly goes into a state of coma, it is fair to surmise that we are confronted with a case of cerebro-spinal meningitis. Should retraction of the head develop, the diagnosis is

almost certain. We must, however, not forget that *pneumonia* may begin precipitately with marked cerebral symptoms. Careful exploration of the chest will decide the question under these circumstances. When pneumonia complicates cerebro-spinal meningitis the pulmonary symptoms do not develop until later in the disease.

Osler has called attention to cases of *typhoid fever* beginning abruptly with delirium, headache, retraction of the head and high fever. Goodno corroborates this observation. If such a case dies early, differentiation is impossible unless fluid containing the meningococcus can be obtained from the spinal canal.

Differential diagnosis rests mainly between cerebro-spinal and *tuberculous meningitis*. In cerebro-spinal meningitis the onset is more sudden; the fever is higher; retraction of the head occurs earlier and is more marked and the nervous manifestations are more irritative and less inclined to become paralytic in nature. The hyperæsthesia of the skin and the petechial rash when present are strong, confirmatory symptoms. Then again, the presence of an epidemic is to be taken into consideration and in the case of tuberculous meningitis the family history, the diathesis and the presence of a tuberculous lesion of the lungs, bones or glands are valuable data. Lastly, *lumbar puncture* remains to be mentioned as the most positive diagnostic method (see p. 458).

**Posterior Basic Meningitis.**—A sporadic form of meningitis, simulating tuberculous meningitis in many respects, has been shown by Koplik (*Amer. Jour. Med. Science*, Feb., '05) to be due to the meningococcus (see p. 454, Tuberculous Meningitis).

**Treatment.**—The treatment of cerebro-spinal meningitis by hot baths has given promising results. They are indicated in the beginning of the disease and undoubtedly exert a sedative effect upon the nervous manifestation. Beginning with a temperature of 98° F. the heat can be increased daily by a degree, up to 105° F. One bath daily is sufficient, and as little handling of the patient as possible is to be advised on ac-



count of the suffering caused thereby. An ice-bag to the head sometimes gives relief from the intense headache.

Of the greatest importance is the feeding of these cases. Extreme emaciation results unless we take advantage of every opportunity of getting sufficient nourishment into the child.

The removal of the exudate by means of *lumbar puncture* often gives relief both of pain and of the pressure symptoms. If exudate is abundant it will prove of benefit to tap the spinal canal every two or three days.

**Remedies.**—As the symptoms are due to the inflammatory process and the presence of the exudate rather than to toxæmia, in this respect differing from most of the other infectious diseases, the remedies that will suggest themselves in the treatment of cerebro-spinal fever are such as exert a notable influence over inflammation. In the early stages *Belladonna* is by far the most valuable remedy, corresponding to the meningeal congestion both symptomatically and pathologically. As soon as exudation develops, *Bryonia*, *Apis mellifica* or *Cuprum aceticum* become indicated. When toxic symptoms predominate over inflammatory, such remedies as *Hyoscyamus*, *Opium* and *Helleborus* are more suitable. Cases with marked petechial eruption and of protracted character call for *Arsenicum*, the snake venoms, and especially *Rhus toxicodendron*. Cases with convulsions call for *Cicuta virosa*.

*Actea racemosa* is useful for the pains and spasms persisting after the acute symptoms have subsided (SEARLE). There is intense occipital headache, like a bolt being driven from the nape of the neck to the vertex, felt with every pulse-beat; stiffness of neck; delirium.

*Apis mellifica*.—Sopor, interrupted by piercing shrieks; squinting; pupils dilated; retraction of head (stage of effusion).

*Arsenicum*.—Protracted and adynamic cases; intermittent type.

*Belladonna*.—High fever; convulsions; flushed face; photophobia; difficulty in swallowing; intense throbbing headache; delirium; vomiting; marked drowsiness.



*Bryonia*.—Bursting headache; apathy; child cries when it is touched or moved; arthritis or pneumonia.

*Camphora*.—Fulminating cases with collapsic symptoms.

*Cicuta*.—The toxicologic reports of this remedy show its pronounced action upon the meninges of the brain and cord, in which it sets up intense congestion with resulting *convulsions*. Various forms of paralysis may follow upon the convulsions. More or less disturbance of consciousness is associated. Dr. Baker (*Trans. New York Hom. Soc.*, 1872) reported most promising results from *Cicuta* after using it in an epidemic at Batavia, New York.

*Cuprum aceticum* has long been recognized as a potent remedy in meningitis, and was used with success by Dr. George Schmidt, of Vienna, for the cerebral symptoms accompanying the infectious diseases. Goodno considers *Cuprum aceticum* the most generally useful remedy in cerebro-spinal meningitis, giving it the preference over *Cicuta* when cerebral symptoms predominate over the convulsive symptoms. *Crotalus* has been recommended in cases with marked blood changes.

*Gelsemium*.—Early stages, chilliness, aching and prostration, photophobia, ptosis and squinting; occipital headache, with muscular soreness in the neck; remitting fever.

*Helleborus*.—Stupefaction, child bores its head into the pillow; suppression of urine, convulsions (serous effusion).

*Hyoscyamus*.—Muttering or wild delirium, unconsciousness, convulsions, pupils dilated, purplish rash.

*Kali hydrojod.*—Iodide of potash is the remedy chiefly relied upon by the old school. There are some encouraging reports from its use, and it may be tried with advantage in cases not presenting marked symptoms for another remedy.

*Opium*.—Deep coma, pupils fixed, stertorous breathing, pulse irregular, inclined to be slow, clammy skin.

*Rhus toxicodendron*.—Petechial form, patient restless, profoundly prostrated; herpetic and purpuric eruptions, intense aching pains in back and extremities, tongue dry and brown with reddish tip.

*Veratrum viride*. — Fulminating cases with convulsions, furious delirium, intense cerebral congestion and splitting headache, vomiting, double vision, numbness of limbs and flying pains ; pneumonia.

Some of the *sequelæ* may be relieved by the following remedies:

Neurasthenia, loss of memory, vertigo—*Argentum nitricum*, *Cannabis Indica*, *Cocculus*.

Neuralgia—*Actea racemosa*, *Gelsemium*, *Zinc. phos.*

Ocular symptoms—*Gelsemium* (serous choroiditis, paralytic squint), *Phosphorus* (optic neuritis), *Strychnia*.

The *deafness* following cerebro-spinal meningitis is usually looked upon as hopeless, but Dr. Searle, of Brooklyn, has reported good results from the use of *Silica* and *Sulphur*.

*Diphtheria antitoxin* has been recommended recently by a number of writers, but the results have in no way been uniform. Peabody (*New York Med. Record*, May 13, '05) tried this form of treatment in twenty-two cases, with a resulting mortality of 50 per cent. He says: "It is fair to assure you that there has not seemed to any of us who have watched these cases any influence for good or evil to be ascribed to the treatment of them by diphtheria antitoxin."

#### MALARIA; MALARIAL FEVER.

Malaria represents a group of febrile affections resulting from infection with micro-organisms belonging to the class of protozoa. Each type of malarial fever is traceable to a distinct variety of micro-organism, possessing its own morphological and biological peculiarities. There is a specific parasite for tertian intermittent fever, for quartan intermittent fever, and for æstivo-autumnal fever, or tropical malaria. These parasites attack the red blood-corpuscles, in which they live and develop to full maturity and sporulation. With the completion of sporulation a malarial paroxysm is always observed. The tertian organism requires forty-eight hours to undergo a complete developmental cycle; consequently a pa-

tient infected with this parasite will experience a paroxysm every third day, *i. e.*, with the occurrence of sporulation. Infection with the quartan parasite results in a paroxysm occurring every fourth day. Double infection with the tertian parasite, each group maturing on separate days, results in daily paroxysms. This is the most frequent type in the acute intermittent fevers in this latitude (OSLER). Quartan fever is extremely rare in this country. This parasite may be present in the blood coincidently with the tertian parasite. By such a combination most puzzling types of fever are produced. The parasite of æstivo-autumnal fever is smaller than the other types of parasite, and is practically confined to the Southern States in this country.

The disease prevails endemically in certain localities, which are known as malarial regions. Although low, swampy and poorly drained regions and the banks of sluggish streams are the most frequent localities for malaria, still it also exists in many of the larger cities, especially in their suburbs and along the river fronts. The disease is conveyed to man by the sting of the mosquito, the genus *anopheles* being the one capable of acting as a host for this parasite. Malaria has no doubt increased in the northern cities since the influx of laborers from the South and from Italy has grown to such proportions.

The *pathological changes* resulting from malarial infection are intense anæmia, due to destruction of the red corpuscles by the parasite; enlargement of the spleen, which may lead to hyperplasia of the same; pigmentation in the liver, kidneys and brain. In cases which have resulted fatally there may be intense pulmonary congestion or pneumonia; nephritis; gastro-enteritis. Fortunately, fatal cases are rare, the pernicious form of malarial fever being quite uncommon in this locality.

**Symptomatology.**—A typical malarial paroxysm, consisting of three well-defined stages, namely, chill, fever, and sweat, is seldom seen in children under six years of age.

Both the first and third stages may be absent or but poorly defined. Instead of a chill there may be only the signs of a vasomotor spasm, such as blueness of the finger-nails, cyanosis of the face, cold extremities and yawning, or there may be vomiting, diarrhoea and even convulsions or a comatose state preceding the accession of fever. In the course of an hour or less the fever rises rapidly and may attain to an alarming height. This condition of hyperpyrexia lasts for an hour or two, ending by a gradual fall. Sweat may be entirely absent after the fever. Instead of even this attempt to simulate a malarial paroxysm as it occurs in the adult, the case may assume more of a remitting type. Holt observed convulsions ushering in the attack in four instances, and in two-thirds of his cases there was vomiting. Sheffield (*N. Y. Med. Jour.*, Oct. 23, 1897) reports a series of cases occurring in New York City, the average age of which was ten years, and in one-third of these the chill was absent. In twenty-one cases there were fifteen of the quotidian type, four tertian, one tertian and quotidian (mixed), and one quartan and quotidian.

When there is a complete remission of fever the child may appear well until the second paroxysm makes its appearance. As these attacks return they become more and more atypical, and the condition may go over into one of a remitting type of fever. If this continues uninterruptedly, grave constitutional symptoms develop, such as prostration; heavily coated tongue; abdominal tenderness; slight jaundice. This is frequently called typho-malarial fever, but there are no grounds for supposing such a condition to be dependent upon a mixed infection with the bacillus of typhoid fever and the parasite of malaria.

Enlargement of the spleen and anæmia are usually well marked, especially if the disease has progressed to any considerable extent. The symptoms accompanying the febrile stage are those common to febrile disturbances in general.

The *prognosis* is usually good. Untreated cases may take one of the following courses: (1) mild cases may go on to spontaneous recovery; (2) the paroxysm may gradually diminish in intensity, but grave anæmia and chronic cachexia develop, or (3) the paroxysm may increase in severity and assume finally a pernicious type (THAYER, *Lectures on Malarial Fever*, 1897).

*Masked or Irregular Forms of Malaria and Malarial Cachexia.*—Malaria is seen in its masked form more frequently in children than in adults, and a malarial paroxysm may be so atypical, or affect a certain region to such a degree, as to entirely mask the condition, the malarial element only being eventually suspected by the regularity of recurrence of the attack, the association of enlarged spleen and anæmia, and possibly by a history of exposure to malarial infection or residence in a malarial district. Finally, the discovery of the parasite in the blood expels all doubt as to the true nature of the case.

Disturbances in the nervous system are common. Headache, continuous or recurring; neuralgia in various localities; intermittent spasmodic torticollis, accompanied by a slight rise in temperature and enlarged spleen (HOIT); multiple neuritis. Trigeminal neuralgia is rare in children. Congestion of the lungs, simulating pneumonia, may occur paroxysmally.

*Malarial cachexia* may develop likewise without malaria having been suspected, either from the attacks being unaccompanied by very high fever, or from presenting themselves in a masked form. The child is markedly anæmic and emaciated, the skin being dry and sallow. The face has a drawn, pinched look, and the eyes are surrounded by dark circles. Indigestion and diarrhœa, irregular febrile movements and enlargement of the spleen are usually present. Here, again, an examination of the blood will corroborate the diagnosis. The prognosis in such cases is not as favorable as in fresh febrile attacks.

**Diagnosis.**—Malarial infection should always be suspected when a periodic disturbance, accompanied by anæmia and enlargement of the spleen, is encountered. In order to remove all question of doubt, a blood examination should be made. A negative result does not necessarily exclude malaria, as it may require several examinations in order to find the plasmodium. Even in the absence of the plasmodium a leucopænia together with an increase in the large mononuclear leucocytes is pathognomonic of malarial infection (see p. 419).

*Anæmia infantum pseudo-leukæmia* presents some of the symptoms of malarial cachexia, but the absence of fever, the leucocytosis and absence of the malarial parasite readily differentiate the two conditions.

The remittent form of malarial fever is frequently confounded with such conditions as the *hectic fever of tuberculosis*; *typhoid fever*, and the *septic fever of empyema, pyelitis*, etc. A careful process of exclusion is therefore necessary in order to justify a diagnosis of malaria in many instances. I would especially warn against neglecting to make a most thorough examination of the chest, as I have seen *sacculated empyema* simulate *intermitting* malarial fever as well as the remitting type. Here leucocytosis is also present, as a rule. The old school attaches great importance to the therapeutic test, *i. e.*, improvement of symptoms upon the administration of *Quinine* and in doubtful cases this is justifiable.

**Treatment.**—Little can be done for the patient during a paroxysm to render him comfortable, but fortunately its duration is not long enough to cause material harm. During the interval and during convalescence a tonic treatment is indicated. Cases simulating typhoid fever are to be managed on the same general principles applying to such cases.

Remedies prescribed in malarial fevers are usually divided into three classes: (a) those possessing a specific and abortive influence over the paroxysms, (b) those indicated for general disturbances arising during and complicating the paroxysm, (c) those indicated in the chronic form and for the cachectic manifestations.

To the first class *Cinchona* and its alkaloid, *Quinine*, belong pre-eminently. We must all admit its specific action in typical cases of malarial fever; and while it is, in general, overestimated and given far too heroically, still it remains the most important remedy for the disease. I have, however, encountered a number of cases in my practice that were not cured by *Quinine* and which were promptly relieved by a remedy selected purely symptomatically.

The true sphere of *Cinchona* lies in that class of cases which presents each stage well marked, with the absence of any complications or symptoms not directly traceable to the febrile paroxysm. *Chininum sulph.* is supposed to exhibit greater regularity in the time of occurrence of the paroxysm, besides possessing some symptoms not found under *Cinchona*. For a fuller description of these remedies and their special indications in intermittent fever I must refer to Allen's *Therapeutics of Intermittent Fever*. As to the dose, that is unfortunately a matter of contention. Kafka (*Homœopatische Therapie*) sums up his experience as follows: "Given on exact indications, *Quinine* acts in small as well as in larger doses, but not in infinitesimal doses. While the most beautiful results were attained with the 1x trituration, or even stronger doses of one to two grains given every two hours during the period of apyrexia, we exerted ourselves in vain with the 2d, 3d, etc." Goodno (*Practice of Medicine*) expresses similar views and he recommends the usual therapeutic dose.

The other remedies, aside from *Cinchona* and its alkaloid, that have occasionally given me positive results are *Nuxvomica*, *Eupatorium perfoliatum*, *Ipecacuanha* and *Natrum muriaticum*.

If strong indications for any one of these remedies are present in a case I give it before prescribing *Quinine*.

Remitting malarial fever suggests *Gelsemium*, *Baptisia* and *Chininum arsenicosum*.

In malarial cachexia *Arsenicum* is the most important remedy.



## TYPHOID FEVER.

Typhoid fever is an acute infectious disease, the specific causative agent being the *bacillus of Eberth*. This germ is found abundantly in the discharges from the bowels; also in Peyer's patches, the mesenteric glands and in the spleen. The bacilli have also been demonstrated in the circulating blood and in the urine when there was albuminuria. The *anatomical lesions* are inflammation of Peyer's patches and of the solitary follicles in the ileo-cæcal region with tendency to ulceration and enlargement of the spleen. A maculo-papular eruption of rose-colored spots appearing mainly upon the abdomen is one of the pathognomonic signs of typhoid fever, but like ulcerative lesions of the intestines it is not so constantly associated with the disease in children as in adults. The typhoid bacilli have been demonstrated in these spots. The accompanying symptoms are fever of a characteristic type; prostration and disturbances in the nervous system; more or less diarrhoea and wasting. Here again it is not as typical as in adults. The fever is more irregular, remissions are more pronounced and the duration is shorter, as a rule. On account of the absence of pronounced ulceration of the bowel in the second week, the temperature does not show the septic course assumed in adults at this time. This condition, however, is not to be absolutely excluded. The associated symptoms are usually milder and diarrhoea may not appear until in the later stages of the disease. There is, on the other hand, a severe type of typhoid fever occurring in children that may present every unfavorable phase of the disease as it is encountered in the adult, not barring copious hæmorrhages and perforation of the bowels, but as a rule the gravity of these cases depends more upon the degree of toxæmia than upon anatomical lesions.

It is only in recent years that the fact of typhoid fever being a common disease during childhood has been recognized. Many mild cases were looked upon as a *simple continued*

fever, while more pronounced ones received the appellation *infantile remittent fever*, or they were diagnosticated *worm fever*. Some confusion as to the gravity of the disease is still to be detected in the deliberations upon the subject. There are several factors influencing an opinion on this question that must be taken into consideration. In the first place, we must bear in mind that there are two varieties of the disease, the mild and the severe type, and the difference between the two is so great that West ("Lectures on the Diseases of Infancy and Childhood") ventured to divide his discourse on typhoid fever in children into a separate consideration of these varieties. One is therefore likely to judge the disease from which ever variety he has mainly encountered. Again, mortality reports from hospitals and from private practice are much at variance because such cases as are brought to a hospital are usually critically ill and come from the poorer classes, where neglect and poverty have undermined the constitution.

The typhoid bacillus is a rod about  $1\text{-}\mu$  to  $3\text{-}\mu$  in length by  $0.5\text{-}\mu$  to  $0.8\text{-}\mu$  in diameter. The ends are rounded. Any of the ordinary anilin dyes will stain it, and decolorization takes place with Gram's method. Vacuoles and highly refractive bodies at either extremity are observed in bacilli grown on culture media. The vacuoles do not stain; they represent retrograde changes. One of the peculiarities of the bacillus is its motility, which is destroyed by the blood serum of a patient suffering with typhoid fever. The serum also causes agglutination and clumping of the bacilli, indicating that an antitoxic substance has been produced in the blood of the infected person. This is the Widal reaction and it may persist for several years after the attack, *i. e.*, until the immunity runs out. It may be observed as early as the sixth day, but usually not before the eighth.

Typhoid fever is a metastatic infection, the bacilli being distributed in groups throughout the body. Beside giving rise to the lesions seated in the solitary follicles, Peyer's

patches, mesenteric glands and spleen, the typhoid bacillus may also produce pneumonia, meningitis, osteomyelitis, pleural effusion. Under certain conditions it also acts as a pus producer, giving rise to abscess of the spleen and liver. As a rule, however, complications are due to an admixture of other bacteria, the commonest mixed infections being with the streptococcus, staphylococcus and pneumococcus.

Infection takes place through the alimentary tract. The commonest source of infection is drinking water that has been contaminated with the dejecta of typhoid fever patients.

Milk is a common carrier of the infection. As the bacilli grow rapidly in milk, the adulteration of this commodity with contaminated water becomes a grave matter. The possibility of the germ entering the system through the inspired air and the occurrence of some cases by direct transmission from one patient to another is not to be denied, but such cases must be extremely rare. Henoch has seen patients lying beside children with typhoid fever, who discharged their stools directly into the bed, contract the disease, and similar cases of house infection have been reported from the Children's Hospital in Basel, by Hagenbach-Burckhardt and by others. The degree of contagiousness, however, is so slight that it need not be taken into consideration when any degree of sanitary precaution is exercised.

Typhoid fever is rarely encountered before the second year, but there is no doubt that it does occur during infancy. A number of authentic cases are on record, and I have personally encountered it. In two reported epidemics of wide distribution 1 per cent. of the cases occurred in infants under two years old. Samuels (*New York Med. Journal*, July 28, 1900) reports a case in a child eighteen months old; the diagnosis was verified by the Widal reaction and the finding of Eberth's bacillus in the stools. The fever continued for twenty days. There was no rash or diarrhoea. Recently I saw a case in an infant one year old with Dr. Bellville. There were rose spots, diarrhoea, enlarged spleen, continued

fever, lasting twenty-one days. The Widal reaction was present. Among ninety-seven cases observed by Henoch two were in infants under one year; twenty-one from the second to fifth year; fifty-nine from the fifth to tenth year. Von Steffens found among one hundred and forty-eight cases two under one year (BAGINSKY). The majority of cases are seen after the sixth year. Boys are more frequently attacked than girls. Epidemics are more prevalent in the fall than at other seasons. In this climate September and October furnish the largest number of cases, although in many large cities the disease is practically endemic. In some epidemics children are more affected than adults, as occurred here in a recent spring epidemic.

**Pathology.**—The *pathologic lesions* are not as marked as in the adult for anatomical reasons. The first change observed in the intestines is a catarrhal inflammation of the lower portion of the ileum, together with swelling of the solitary follicles and Peyer's patches in the ileo-cæcal region. The cæcum and colon are moderately involved in the catarrhal inflammation.

As the process continues round-cell infiltration into the lymphoid structure constituting the swollen follicles and patches takes place, with the formation of elevated plaques and shot-like projections. The amount of infiltration, however, seldom attains to the degree observed in the adult, and, instead of necrosis from compression of the blood-vessels supplying the affected area setting in, it usually terminates by fatty degeneration and resorption of the infiltration. For this reason the course is shorter and more benign, and ulceration of the bowels is much rarer than in adults. In older children, however, the same lesions are to be found that characterize typhoid fever in adults. With the breaking down of the infiltrated areas, deep oval ulcers, their long axis corresponding to the direction of the bowel, are found. Smaller, irregularly-scattered ulcers result with the breaking down of the solitary follicles. The slough is more frequently superficial, separat-

ing without the production of a deep ulcer and unattended by the septic fever observed in adults at this stage. Grave symptoms are more frequently dependent upon toxæmia than upon anatomical lesions. General infection without localization is also possible.

The changes found in other parts are swelling of the mesenteric glands, swelling of the spleen, which is soft and pulpy; parenchymatous degeneration of the heart, liver and kidneys. Hypostatic pneumonia, bronchitis of the finer tubes and broncho-pneumonia are commonly associated with typhoid fever. These lesions are usually due to a secondary infection, the typhoid bacillus only rarely producing them (see above). Slight pathological changes in the kidneys are common, and severe lesions may occur. Bacilli are present in the urine in about 20 per cent. of cases during the third and fourth week (PARK), and the urine may become cloudy from their presence.

**Symptomatology.**—The onset of typhoid fever is gradual in the majority of cases, being preceded for a day or two by prodromal manifestation, such as general malaise; headache; restless and dream-disturbed sleep; anorexia and constipation. There may be slight chilliness recurring for several days, but rarely a decided initial chill. The temperature now begins to rise in a characteristic manner. Morning remissions are marked, but the fever rapidly reaches its acme, usually in from four to five days; in adults this is not attained until the end of the first week, and there is a more gradual step-like rise in the temperature.

At times the temperature rises abruptly instead of ascending gradually. This is more common in children than in adults. The temperature soon reaches its maximum evening rise ( $103^{\circ}$  to  $104^{\circ}$  F.) and by the end of the second week a rapid decline in the temperature is the rule in such cases. On the other hand, an abrupt beginning with a high fever ( $105^{\circ}$ ) and early delirium is characteristic of the gravest (fulminating) form of the disease, namely, *acute typhoid septi-*

*cæmia*, and it may not be possible to decide at once whether this type or a milder type with a complication (pulmonary) is confronting us. After the acme has been attained the fever presents a continuous remitting type. The remission occurs in the morning, and the exacerbation in the evening; in severe forms, with high temperature, the remissions are not as marked as in milder cases. This is a sign of prognostic importance (see "Prognosis"). Toward the end of the second week (about the twelfth day) the morning remission be-

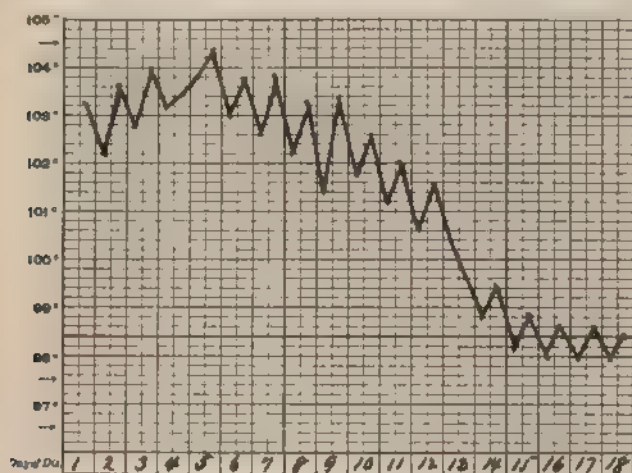


FIG 59. TEMPERATURE CHART FROM A CASE OF TYPHOID FEVER IN A CHILD FOUR YEARS OLD, ILLUSTRATING RAPID ONSET AND SHORT COURSE.

comes more pronounced, and soon a lowering in the evening rise is noticed. The temperature now falls by lysis, and in the course of from a few days to a week the stage of defervescence is completed. Accordingly, a typical, uncomplicated case of moderate severity occurring in a child under ten years old pursues a course of from fifteen to nineteen days. Severe cases, or such in which complications occur, may be indefinitely prolonged beyond this period, or prove fatal.

The age of the child also exerts an influence upon the duration of the fever. In children of five years the average duration is 15.7 days; at eight years, 18.3 days, and at ten years, 20.3 days (Montmollin).

The symptoms occurring during the first stage are fever, accompanied by prostration, gastric derangement and marked indifference. The face is pale, and the cheeks usually flushed; later, the countenance wears a characteristic apathetic expression, and assumes a sub-cyanotic color, due to the impaired surface circulation.

The *temperature* is not always an index of the severity of the infection. While abrupt onset with early hyperpyrexia indicates an intense infection, still a weak heart and low vitality may be the reason for failure to react against the disease and consequently we may have most serious cases with but moderately high fever. Cases are on record in which no rise of temperature occurred, and still grave symptoms were present. Jacobi (*Pediatrics*, Dec., 1899) believes that the very severe typhoids that exhibit bad cerebral symptoms are likely to have low temperatures on account of the thorough sepsis prevailing. In such the prognosis is bad. A sudden rise of temperature during the course of the fever usually indicates a complication, while a sudden fall means hæmorrhage or perforation.

The *tongue* is heavily coated with a light-yellowish fur. This coating wears off in places exposing the slightly swollen papillæ as red specks. A red streak down the centre is likewise produced by the tongue rubbing against the upper central incisors during its propulsion. Only in severe and protracted cases does the tongue become brown and cracked. Even when the lips are covered with brown crusts and cracked and bleeding, the tongue is often more or less moist and light in color.

Cracking and bleeding of the lips is common in children, from the great prevalence among them of picking at the same. The mouth is dry, the tongue being heavily coated,



and the breath offensive. Aphthæ and thrush do not appear nearly so frequently in typhoid as in ileo-colitis.

The *bowels* are usually constipated in the beginning, but they may become loose as the fever progresses. In cases marked by severe bowel symptoms the stools are thin and watery; often involuntary. The abdomen is prominent, and tenderness and gurgling are found in the right iliac fossa upon pressure. This is due to the accumulation of fluid in the lower ileum and may be present even when there is constipation. In infants there is considerable mucus in the stools and they conform more to the type found in an ordinary catarrhal inflammation. Gas forms plentifully, but owing to the paretic condition of the gut it is not readily expelled. The typical typhoid stool is of a dirty, yellow color and thin, even consistency, being appropriately described as the "pea-soup stool." It has a characteristic penetrating offensive odor, which may cling stubbornly to the patient. During the second week when delirium sets in the stool is usually involuntary; this condition may remain to the end in adynamic cases.

The *eruption* is not so constant and is less abundant in young children than in adults; it is found upon the abdomen and lower portion of the chest, developing in crops. It is absent in perhaps 20 per cent. of cases (JACOBI). The first crop appears about the eighth day, successive crops appearing for a week or longer. The spots consist of small, rose-colored macules, disappearing on pressure. They may spread to the neck and lower extremities, and in serious cases with septic infection petechiæ may develop. In Dr. Bellville's case, an infant one year old, the spots were on the neck and face as well as on the abdomen and chest.

The *spleen* becomes enlarged early in the disease; in fact, by the end of the first week it can usually be felt at the border of the ribs. It may serve as an index to the progress of the disease, its return to normal size during the middle of the third week auguring a good prognosis. If it fails to diminish in size there will be a relapse (JACOBI).

The *pulse* furnishes valuable data for diagnosis early in the disease. Instead of rising progressively with the temperature during the first week of the fever, it remains slow and measured. With the progress of the fever, however, it becomes rapid and feeble. We should, therefore, always suspect enteric fever whenever a febrile condition is encountered in children in association with a relatively slow pulse-rate in the early stage. The opposite condition holds good in meningitis. The dicrotic pulse, so characteristic in adults, is observed only in older children.

The disturbances of the *nervous system* are apathy, prostration and cerebral irritability. The child is often exceedingly cross and slow in answering questions and obeying requests. Delirium is usually present, especially during the night, and if the child is particularly susceptible to the typhoid poison, symptoms resembling meningitis may develop. Thus, dilated pupils, retraction of the head, twitching of the muscles of the face and extremities, crying out in sleep and stupor are frequently encountered. They disappear with the fall in the temperature. The toxic irritation of the brain sometimes produces choreiform movements, such as are observed in chorea gravis. The prognosis is grave in these cases. A true cerebro-spinal meningitis may complicate typhoid fever in rare instances.

The *urine* may become albuminous from acute parenchymatous degeneration of the kidneys. The bacillus is usually present in the urine in such cases, which probably accounts for the albuminuria. Actual nephritis is rare.

The *blood* undergoes no important changes in the early stages of the disease, but by the third week a decided anæmia has developed, due to a reduction both in the number of red corpuscles and in the amount of hæmoglobin (THAYER). Leucocytosis does not appear unless perforation or secondary infection occurs.

Besides the rose-spots, sudamina frequently develop upon the skin, mainly on the chest and abdomen. They appear in

the later stages of the disease. At this period profuse and debilitating sweats may occur, with subnormal temperature. The patient seems collapsed after the sweats, but I have never seen cause for alarm from this condition, although a stimulant is generally necessary to bring the temperature back to normal. It is quite customary to find a subnormal morning temperature continuing for some time during convalescence.

Bed-sores, boils, phlebitis and abscesses in various parts are seen in *septic cases* and in the debilitated.

*Abortive Type.*—Instead of running its full course typhoid fever may abort at any stage. Such a case begins as an ordinary attack and there may be all of the characteristic symptoms, *i. e.*, nosebleed; ascending fever; iliac tenderness; dry, coated tongue and rose spots, but by the tenth or twelfth day the temperature will have returned to normal and convalescence be in progress. Formerly the term, *simple continued fever* was applied to this class of cases, but it is doubtful if a continued fever occurs without an infective agent. I have obtained the Widal reaction in most of my cases of this class going beyond eight days.

*Relapses.*—These may be said to occur in about 10 per cent. of cases. They usually occur during the first two weeks of convalescence, but a sudden rise in the temperature and a return to the original fever curve, together with the reappearance of symptoms, may set in during the latter part of the third week before the evening temperature has yet become normal. A relapse indicates a reinfection with germs that have escaped destruction and it is accompanied by the symptoms of the original attack. A fresh crop of rose spots usually appears. The average duration is from ten to fourteen days. The symptoms are usually mild, but death may occur during a relapse. This is often the case when a relapse is brought on by a premature return to solid food. Baginsky believes children to be especially prone to relapses. I believe them to be more frequent in certain epidemics.

Among the complications, bronchitis and broncho-pneu-

monia are the most frequent. *Bronchitis* is almost a constant accompaniment of typhoid fever. *Broncho-pneumonia* is not uncommon ; this complication is a frequent immediate cause of death in the grave types of the disease. A *lobar pneumonia* may also complicate typhoid fever. This may be due to a mixed infection with the pneumococcus or it may be due primarily to the typhoid bacillus. While not altogether characteristic, still in this form of pneumonia, as a rule, the hæmorrhagic element of the pulmonary consolidation is prominent. The sputum is also markedly hæmorrhagic, not unlike that observed in pulmonary infarct. In one of my cases of typhoid fever there developed during the second week a large area of pulmonary consolidation, and there was a copious, deep-red, jelly-like expectoration. This expectoration contained typhoid bacilli in large numbers. Sappington (*Hahnemann Hospital Bulletin*, Dec., 1905) reports a typical case of typhoid pneumonia in a child seven years old in which the diagnosis was verified by post-mortem blood cultures. He calls attention to the fact that in these cases leucopænia is more likely to be present than a leucocytosis, even when the pneumonic process is due to a secondary infection of the lungs with the pneumococcus. *Otitis media* ; *bedsores* ; *circumscribed suppurative processes* ; *phlebitis* and *intestinal hæmorrhages* are occasionally seen. Fatal hæmorrhages and perforation are rare, but perhaps not so rare as is generally supposed. *Abscess of the lung*, *empyema* and *septic parotitis* are also among the rare complications, usually seen only in hospital practice. They are almost always fatal.

*Hæmorrhage* occurs most frequently during the third week. Its indications are collapse and a rapid fall of the temperature. Death may occur before blood is expelled. *Perforation* is less common than hæmorrhage and presents the most serious of all accidents. Characteristically it is preceded by sharp abdominal pain followed by collapse, and usually intestinal hæmorrhage. The condition, however, may be masked and not suspected until peritonitis develops.

Other conditions that have been found associated are ulceration of the mouth, throat, and genitals; peri- and endocarditis; peritonitis; suppurative synovitis and osteitis; nephritis; tuberculosis.

*Sequelæ affecting the nervous system* are transitory aphasia; multiple neuritis; chorea and insanity, all fortunately rare.

**Prognosis.**—The prognosis is, on the whole, more favorable in children than in adults. Perhaps the chief reason for this is the average shorter duration of the fever, the greater tolerance on the part of the heart and the lesser liability of severe hæmorrhage and intestinal perforation, but we must bear in mind that the previous health of the child and the development of one of the graver complications must be carefully considered in estimating the prognosis. In young infants the prognosis is grave. The mortality rate is not very uniform, thus Holt has placed it at 5.4 per cent.; Steffen at 6.7 per cent.; Henoch at 7.5 per cent. and Baginsky at 9 per cent. Judging from my own experience and from observation of the practice of my colleagues it is considerably lower under homœopathic treatment combined with rational hydrotherapy.

The age is an important factor; the intermediate ages are the most favorable (ROEMHELD). I recall hearing an eminent clinician remark at the bedside of a child nine years old, "It is, indeed, fortunate to have typhoid fever when you are nine years of age." The pulse and temperature are ordinarily a safe guide, but as stated above the amount of fever does not always indicate the degree of infection. It is, therefore, best to go direct to the heart, auscultating daily to ascertain the condition of the heart muscle. When the *pulse-rate* remains relatively low in comparison with the fever and its volume is good there is no immediate danger to be feared. A rapid pulse, especially when this occurs early in the disease, is an unfavorable omen.

Regarding the *temperature*, the absolute height of the fever in uncomplicated cases is of prime prognostic importance. "With every day that the temperature retains its high range

without interruption the danger to the patient grows." (KLEMPERER). We can usually judge of the course that the fever is about to run after we have observed the case up to the end of the first week. It is rare for the temperature to rise above the point attained at this time. The duration of the fever in cases of abrupt onset with high fever is, as a rule, short. This does not, however, apply to fulminating typhoid (see above). The daily variations in the fever are also important prognostic points; the greater the daily remissions, the less destructive to the organism will be the fever, while a continuously high fever with but slight diurnal variation, and one that is not influenced by baths, etc., offers an unfavorable prognosis (KLEMPERER, *Modern Clinical Medicine*, 1905).

*Complications*, such as pneumonia, septic infection, hæmorrhage and tympanitis, always render the prognosis more unfavorable. In the fatal cases coming under my notice there was present, as a rule, a grave secondary infectious condition, such as septic parotitis, empyema, pulmonary abscess and osteomyelitis. Acute typhoid septicæmia is also fatal in the majority of instances. Geohegan (*Trans. Amer. Institute of Hom.*, 1897) reports a fatal case from perforation and hæmorrhage in a child under two and a half years old. Ratier, of Paris, reports twenty-two cases of hæmorrhages in a series of seven hundred and sixty-two cases, ten of which resulted fatally.

**Diagnosis.**—Aside from the pathognomonic symptoms of typhoid fever, viz., continued fever of a definite type, rose-colored spots, tympanitis with gurgling and tenderness in the right iliac fossa, enlarged spleen and pea-soup stools, there is at our command the blood test of Widal and the urinary test (diazo-reaction) of Ehrlich. Unfortunately for the general practitioner, the former is difficult to carry out, requiring special laboratory facilities and expert technique in bacteriology. In every large city, however, there are pathological laboratories where this test can be made so that it is rarely necessary for the physician to be especially equipped. Ficker has devised a

substitute for the Widal method in the form of a glycerine emulsion of typhoid bacilli. This has been placed on the market as Ficker's Diagnosticum and should prove of great practical value to the practitioner. Widal's test consists of the introduction of a few drops of blood from a patient suffering with typhoid fever into a pure culture of typhoid bacilli. A microscopical examination reveals a prompt formation of clumps consisting of the agglutinated bacilli, which have also lost their motility. The reaction is one of infection and of immunity, indicating that a toxic substance has been formed in the blood serum, which is capable of destroying the motility of the germs causing the disease, and also inducing their agglutination. Johnson (*Amer. Public Health Assoc.*, 1896) advocated the use of dried-blood specimens as more expedient, and this method is now largely employed. By simply redissolving the dried blood, which has been collected upon a piece of unglazed paper, with a little water and adding this solution (1 to 40 or 50 dilution) to an equal quantity of a young bouillon culture of typhoid fever bacilli, the reaction is obtained just as satisfactorily as with the fresh blood. The reaction may be observed on the fourth day of the disease, but it is usually delayed to the end of the first week. It continues throughout the fever and may persist for some time after the recovery. The frequently-recorded negative results should not weigh heavily against this most valuable diagnostic adjuvant, as faulty technique is probably more to be blamed than the test itself. The proportion of cases in which a definite reaction occurs and the time of its appearance, based on an extended Health Department Laboratory experience, is given by Park (*Bacteriology*), as follows: 20 per cent. gave positive results the first week, 60 per cent. in the second week, 80 per cent. in the third week, 90 per cent. in the fourth week. In 88 per cent. of the cases in which repeated examinations were made (hospital cases) the reaction was found at some time during the fever. Withington (*Boston Med. and Surg. Jour.*, May, 1901) reports two



hundred and fifty-three cases, with but 4 per cent. failures. Its late appearance, usually not before the eighth day, renders it less valuable as an early sign.

The *diazo-reaction* is a valuable corroborative test, but it is also obtained in acute miliary tuberculosis and in rapidly progressing pulmonary tuberculosis. In fact, a large number of infectious conditions will give this reaction, notably measles. I also obtained it in a case of suppurative adenitis. For this reason it is not so conclusive as was first supposed. It is said to be absent in diphtheria. The reaction is a rose-red color imparted to the urine by the addition of ammonia after the urine has been treated with Sulphanilic acid and Sodium nitrite. It is present from the middle of the first week until the end of the fever period; the presence of nephritis interferes with this action. I have been impressed with the large number of cases one encounters in which the clinical picture presents nothing more than a continued fever of remitting type, anorexia, a heavily-coated tongue and constipation. Gurgling in the ileo-cæcal region and tenderness are usually so ill-defined that they are easily overlooked. Rose spots and enlarged spleen may be absent. And yet, no other diagnosis than typhoid infection is to be thought of, which is eventually corroborated by the Widal reaction in the majority of these cases.

Another aid in the diagnosis of typhoid fever in children is the presence of an *epidemic*. Cases occurring sporadically may present difficulties. In all cases of simple continued fever occurring during an epidemic the Widal test should be made. Sahli, of Bern, during an epidemic, obtained it in a number of cases that were sick for only a few days.

Cases without intestinal localization will present difficulties in diagnosis. In such, only a bacteriological examination of the blood will solve the problem. Many of the acute typhoid septicæmias are of this character. They present the picture of a profound toxæmia with high fever and early delirium. Death may occur before it is possible to reach a diagnosis, and the post-

mortem findings may be entirely negative (OSLER, *New York Med. Jour.*, Nov., 1899). In such cases I think we are justified in diagnosing typhoid fever, if we are unable to demonstrate a lobar pneumonia, or if meningitis can be excluded.

From *malarial fever* it is to be differentiated by means of a blood examination to ascertain the presence or absence of the malarial parasite, and by the temperature curve.

*Meningitis*.—A strong point of difference between meningitis and typhoid fever is the behavior of the pulse. In typhoid fever it is relatively slow in the beginning, becoming rapid toward the end of the disease; in meningitis the pulse rises proportionately with the fever in the beginning, but becomes slow and irregular towards the close of the case. Furthermore, in meningitis the abdomen is retracted, the bowels are constipated throughout, and paralyses of the cranial nerves are to be observed. The reflexes are exaggerated, and Kernig's sign may be elicited. None of these symptoms are present in typhoid fever. Meningeal irritation is common, but true meningitis is very uncommon. In typhoid fever of the cerebro-spinal type, it may be necessary to resort to lumbar puncture before a positive diagnosis can be made.

*Acute miliary tuberculosis* may present difficulties in differential diagnosis. Aside from the absence of the Widal reaction in tuberculosis there is a more rapid pulse and greater irregularity in the course of the fever. Often the "inverted type" of fever is noted. Aside from this there is rapid breathing and dyspnoea when the lungs are involved, together with pronounced catarrh of the finer tubes. An old tuberculous lesion may be demonstrable. True meningitis is commonly associated. The most difficult cases to differentiate are those in which abdominal symptoms are the predominating feature.

Early pronounced localization of the infection in some other system than the intestinal tract may lead to confusion. As Osler (*loc. cit.*) emphasizes, the brunt of a very acute infection may fall upon the cerebro-spinal, the pulmonary, or the

renal system. Such cases would be more appropriately designated "typhoid infection" than "typhoid fever."

**Treatment.**—The patient should be put to bed at once in a room that can be freely ventilated, and from which all unnecessary furniture and draperies have been removed, not especially on account of any degree of contagiousness on the part of the fever, but in order to give as much air-space as possible and make it less difficult to keep the room clean.

Provision must be made for the disinfection of the stools and urine, which can be accomplished by the use of an active germicide. A strong solution of chloride of lime, Platt's chlorides, or carbolic acid (5 per cent. solution) is to be poured over the stools as soon as they are passed, and allowed to act upon them for several hours before they are emptied into the water-closet. All towels, napkins and sheets soiled by the patient should be boiled in order to render them sterile.

The *diet* is of the greatest importance. Owing to the intestinal lesions, solid food must be withheld until at least a week after disappearance of the fever, diarrhœa and abdominal tenderness. Where abdominal symptoms have been pronounced during the fever, it is better to wait even longer before resuming solid food. In the milder class of cases we may return to semi-solid food on the fifth day after the temperature has ceased to rise above 100° F., gradually returning to solid food. Such articles of diet as thoroughly cooked cereals; poached eggs; milk toast; the soft portion of a baked apple; baked potato, etc., should be selected at this time.

Although *milk* is looked upon as an ideal liquid food, still it does not act as such in many cases, and, when given unmodified, may pass through the bowels in firm curds. The stool should, therefore, always be inspected when administering milk, as such curds may induce most unfavorable symptoms. A notable ill-effect of milk observed in some patients is tympanitis; this promptly disappears when the milk is discontinued. In young children it is always best to dilute the milk with barley-water, or administer it predigested.

Strained vegetable soup, made with mutton and a variety of fresh vegetables, is a most valuable food, and an agreeable change to the patient. Likewise grape juice, when diarrhoea is not prominent; and any of the reliable proprietary foods such as Horlick's Malted Milk, Eskay's food, and Mellin's food (the latter when there is constipation), are all of value. The mistake, however, is to feed the patient without any degree of regularity or restriction as to quantity, changing from one article to another promiscuously. The best results are obtained by selecting the food best adapted to the case, and administering three to four ounces every three hours. Some variation in the character of the diet is most agreeable to the patient and a great aid in keeping up the nutrition. The carbohydrates are especially valuable in preventing the marked emaciation so prone to occur in typhoid fever. The patient should also receive water freely.

The child must be sponged daily with cold or tepid water and alcohol (one part to four of water), and when the fever runs high, remaining above 103 degrees F. during the greater period of the twenty-four-hour range, these baths may be repeated every three hours. Should this fail to control the fever, a cold cheese-cloth pack may be tried, or, what I prefer, sprinkling the body with water at 70 to 80 degrees F., and at the same time applying friction to the extremities. In order to carry out this procedure, the child is stripped and laid on an oil cloth sheet over which a linen sheet is spread, while the head of the bed is elevated so that the water may run off into a bucket. The water may be poured from a watering-can or it may be freely squeezed from a large sponge. Sometimes rubbing the body briskly with pieces of ice wrapped in a towel will have a most grateful and beneficial action in cases of hyperpyrexia. I have found these methods a good substitute for the full bath (see p. 18). Should the patient react poorly after any form of cold water treatment it is better to desist and use milder measures (luke-warm sponging).

*The Indications for the Different Hydrotherapeutic Measures*

*in Typhoid Fever.*—It is a mistake to attempt to treat all cases of typhoid fever on the same plan, and by “hydrotherapy” to understand simply the cold bath treatment. While the majority of cases are eminently suited to this mode of treatment in one of its forms, *i. e.*, the full bath, the cold sponge bath, or the cold shower bath, still there are those in which the baths fail either to improve the case or actually aggravate the condition. In such the ice-bag may do good, or intestinal irrigation with luke-warm water (enteroclysis) may be the therapeutic measure indicated. The explanation for this apparent disparity is as follows: Where the brunt of the infection falls upon the vasomotor centres and the pulse is weak, rapid and dicrotic and the peripheral circulation poor (vasomotor-paresis), the intermitting cutaneous shock produced by the cold bath is the form of treatment indicated; where the localization is primarily intestinal and there is intense inflammatory reaction and tendency to hæmorrhage and perforation the ice-bag is useful while the bath may do harm, and where toxæmia predominates and the cerebro-spinal nervous system and the kidneys are chiefly attacked, enteroclysis, by favoring elimination and lowering temperature, is the form of treatment that will do the most good. Some patients present such a pronounced idiosyncrasy against cold water or are in such a condition in which reaction is impossible, that it becomes positively harmful to persist in giving them cold baths or cold sponging. Here luke-warm water, especially if it be allowed to evaporate from the body and in that manner abstract heat, is beneficial and should be resorted to. In hyperpyrexia in children who do not stand cold water, sponging with hot water, especially along the spine, has often proved beneficial.

*Stimulation* may become necessary in the later stages of the fever. The first and most prominent indication is cardiac weakness. Daily auscultation of the heart should be practiced and when the first sound loses its muscular element and resembles the second sound (embryocardia) alco-

holic stimulation should be resorted to. Other indications are continuous delirium of the low muttering variety; dry, trembling tongue; tympanitis and pronounced adynamia, and I may add lack of reaction to remedies. Many of our remedies act prominently in a stimulating way, but we meet cases in which the system fails to respond to them until reaction has been brought about by physiological means. A teaspoonful of whiskey diluted with water may be given every two to three hours to be decreased or increased according to circumstances. Collapse will call for strychnia.

Hæmorrhage, if slight, requires nothing more than temporary withdrawal of food followed by greater caution in feeding, absolute quiet of the patient and possibly a change of remedy. When severe, it proves a grave complication. A cold application to the abdomen in the form of Leiter's tubes or an ice bag will prove of great benefit. Absolute rest must be enjoined, even the bed pan may be put aside and clothes used to collect the excreta. If collapse threatens, stimulants must be used. They should, however, be used cautiously, as overstimulation of the heart may favor increased hæmorrhage. *Strychnia nitrate*, hypodermically, is perhaps the best stimulant to employ. *Geranium* tincture, per rectum, as recommended by Dr. Woodward (*Eclectic Med. Jour.*, June, 1901) using two ounces of tincture in a pint of milk and water, seems a most valuable adjuvant. Infusion of a normal saline solution where loss of blood was great has saved life.

Perforation and peritonitis are extremely fatal complications, although early laparotomy in perforation before peritonitis has set in offers better hope for the patient than conservatism according to the observations of Finney and Keen.

Wescott collected eighty-three well authenticated cases of perforation that were operated, of which number sixteen recovered, making a mortality of 80.6 per cent. Comparing this with Murchison's figures of 90 per cent. to 95 per cent. mortality among unoperated cases, operative interference



seems to offer much for the future. Five of these cases were in children under fifteen years, of which two recovered. The most favorable time to operate has been the second twelve hours after perforation, but under certain circumstances it may be more prudent to operate earlier. Operation should, however, always be deferred until the primary shock has worn off. (KEEN, *Surgical Complications and Sequels of Typhoid Fever.*)

The leading typhoid fever remedies are *Baptisia*, *Bryonia*, *Gelsemium* and *Rhus tox.*, and in certain epidemics one of these remedies may be indicated in almost every case. The selected remedy should be continued throughout the entire course of the disease unless positive indications for a change of remedy present themselves; even in such an event it is wise to return to the first remedy when the intercurrent has corrected the symptoms for which it was chosen. Thus, in many epidemics *Bryonia* will be found the chief remedy, and although indications for *Hyoscyamus*, *Phosphoric acid*, or some other remedy of a similar sphere of action may arise during the progress of the case, a return to *Bryonia* may, as a rule, be made with advantage as soon as these symptoms have been controlled.

It is not at all rare to find cases running a short and uncomplicated course receiving but a single remedy during the entire period, providing the remedy has been carefully selected from the beginning. In a disease like typhoid fever, which we may expect to assume a most grave aspect at any moment, we must prescribe with caution and precision from the beginning and only change the remedy after mature deliberation. As to repetition of the dose, it has been my experience, in common with that of many others, that the best results are obtained from a frequent administration of the remedy when symptoms are urgent, lengthening the intervals as soon as improvement is noted.

The following indications embrace the most important symptoms of the leading remedies at our command :



*Agaricus*.—In typhoid fever where the nervous symptoms predominate. Low fever, tremulous tongue, and general tremor of the entire body. Among adults it is recommended for drunkards in whom the heart is giving out. Alcoholic stimulants must, of course, not be withheld from such cases. We often encounter boys who smoke cigarettes excessively and whose nervous system is about as wretched as the adult drunkard's. Here *Agaricus* is well indicated.

*Apis*.—Remitting type of fever. Chilliness in afternoon with oppression of breathing; heat without thirst; later, unconsciousness with involuntary stools; dry tongue, which is cracked and covered with aphthæ, difficult to protrude; difficult deglutition; scanty urine; muttering delirium.

*Arnica*.—General stupefaction of the senses; general soreness, bed feels too hard; the sleep is disturbed by anxious dreams; the tongue is red and dry, with a brown streak down the centre; putrid taste in mouth; fetor ex ore; involuntary discharge of fæces and urine; the extremities become cold while the head remains hot; hæmorrhages and bedsores develop.

*Arsenicum*.—Low types of typhoid, usually the later stages in unfavorable cases. Farrington cautions against the early use of *Arsenic* in typhoid fever, and considers it a remedy capable of doing harm unless clearly indicated. It is most useful in the young or aged, or in those debilitated by previous ailments. The general symptoms are so characteristic of *Arsenic*, such as great restlessness, prostration; thirst for small quantities of water; hot, dry skin; general aggravation of all symptoms soon after midnight or noon; cadaverous smell of the discharges as well as of the patient, are all prominent indications for its use. "Its true place is there, where rotten, putrid and cadaverous stools and dry, wooden tongue indicates a degree of disintegration of the vital fluids which *Rhus* no longer can check." (JAHR.)

*Baptisia*.—The well-known mental symptom, the hallucination that the body is dismembered, that certain parts of

the body are double, or that there is a second self in the bed with the patient, is a strong indication for *Baptisia*, although its absence by no means deprives this drug of its usefulness in typhoid fever. *Phosphorus* and *Petroleum* both have similar symptoms. The condition calling for *Baptisia* is characterized by great weariness and a bruised feeling of all the limbs, together with a low type of fever and physical prostration; offensive diarrhœa; breath, sweat and urine are alike offensive; there is dull, stupefying headache; the patient is delirious, sleeps heavily and is aroused with difficulty. The tongue is dry and brown, the conjunctivæ are injected; the face is flushed and presents a besotted expression; exhaustion is marked. *Baptisia* may be indicated early in the disease when the symptoms are intense from the beginning, thus excluding such remedies as *Bryonia* and *Gelsemium*.

*Bryonia*.—*Bryonia* may be indicated at any stage, although its most frequent application will occur during the first stage. The symptoms calling for its selection are very characteristic and prominent—irritability, lassitude, desire to remain quiet and sleep; headache, worse from opening the eyes or moving the head; dryness of the lips, mouth and throat, with thirst for large quantities of water; aching of the limbs, worse from motion; frequent brown, putrid stools; delirium at night and restless sleep, disturbed by dreams of daily affairs; wants to go home; visions when closing the eyes.

*Carbo veg.*—*Carbo vegetabilis* is indicated in extreme cases. It has well been said: "The *Carbo vegetabilis* patient is dying," nevertheless, reaction may take place even in such a serious state as it pictures. Many writers speak very highly of this remedy, but personally I am not able to say what *Carbo vegetabilis* will do, as in such a condition I never fail to resort to stimulation. The picture is a familiar one—progressive stupor; lustreless eyes; with sluggish pupils; Hippocratic countenance; parched tongue; distended abdomen; involuntary diarrhœa; hæmorrhages from the nose, mouth or intestinal tract; cold extremities, the coldness gradually

extending from the feet up to the knees; small, frequent pulse, at times imperceptible; decubitus. The *Carbo vegetabilis* patient is passive, the *Arsenicum* patient restless.

*Gelsemium*.—In the early stages *Gelsemium* is frequently indicated on the symptoms of lassitude, drowsiness, dull headache, with heaviness of the eyelids and photophobia; slow, intermitting pulse, accelerated from slight exertion; blueness of the lips; chilliness up and down the spine; epistaxis; catarrhal condition of the eyes and respiratory tract; diarrhoea.

*Hamamelis*.—Hæmorrhages of dark, fluid blood from the bowels, with great soreness of the abdomen.

*Hyoscyamus*.—The delirium indicating *Hyoscyamus* is characterized by loquacity, obscene actions, or even attempts at violence. The patient picks at the bed-clothes and grasps at flocks in the air, with continual muttering. *Stramonium* is similar, but the loquacity is confined to one subject and the patient is more noisy, often crying out in terror from supposed visions of horrible animals, bugs, and the like, which he sees coming out of the floor, crawling along the ceiling, etc. The automatic movements of the extremities occurring during the delirium are also characteristic in both drugs, but in *Hyoscyamus* they are more jerky and spasmodic. *Hyoscyamus* also has total loss of consciousness, with dry tongue, involuntary stools, subsultus tendinum, dribbling of urine.

*Lachesis*.—The *Lachesis* patient, similar to the condition noted under *Hyoscyamus*, is also loquacious, but he jumps from one subject to another in an incoherent manner; there is stupor, dropping of the lower jaw; dry, red, or blackish tongue which is red at the tip and bleeding, and trembles on being protruded; the stools are horribly offensive, the abdomen sensitive to touch, and all symptoms are more intense after sleep.

*Mercurius*.—The characteristic nocturnal aggravation, the greenish-yellow stools, broad, flabby tongue and drowsiness may indicate *Mercurius*, especially when there is hepatic disturbance in connection with the case.

*Muriatic acid*.—Low types of typhoid fever, in which the patient is stupid, sliding down to the foot of the bed; the tongue is parched and dry, difficult to protrude; stools involuntary while passing urine; loud moaning during sleep, and when awake not fully conscious of his surroundings.

*Opium*.—Either complete loss of consciousness with loud, stertorous breathing, contracted pupils, face dark red and bloated or pale with death-like expression, dropping of the lower jaw, hot sweat, or delirium with sleeplessness due to hyperæsthesia of the special senses, so that slight noises keep him awake.

*Phosphoric acid*.—Low typhoid state, in which the patient becomes totally indifferent to his surroundings. He can be aroused, but with difficulty, and soon relapses into his apathetic condition. There is great debility, rattling of mucus in the chest, rumbling in the abdomen, tympanitis, grayish watery stools, bleeding from the nose, red streak through the centre of the tongue, milky urine, clammy skin.

*Rhus tox.*—After *Bryonia* and *Gelsemium*, *Rhus toxicodendron* and *Baptisia* frequently follow. The provings of *Rhus tox.* present a typical typhoid state, and the anatomical changes in the intestines closely correspond to the lesions of typhoid fever. The symptoms are sharp and well-defined, as is the case with *Bryonia*. The mind becomes beclouded and the mental operations are performed with difficulty. The patient is restless from a distressing aching in every limb, and constantly changes his position to gain relief (not as in *Arnica*, where there is soreness produced by lying in one particular attitude, which makes him seek a new position). The sleep is restless, disturbed by dreams of great physical exertion. The lips are brown and dry, and the teeth are covered with sordes; the tongue is likewise brown and dry, presenting a triangular red tip. The diarrhœa is worse during the night, often involuntary during sleep. Beside this, there may be bronchitis, hypostatic pneumonia with bloody expectoration, and bleeding from the nose. Active irritative symptoms

referable to the cerebro-spinal system indicating profound typhoid toxæmia often yield better to *Rhus tox.* than to such remedies as *Hyoscyamus*, *Helleborus* and *Stramonium*. It is not always possible to get clear-cut indications upon which to differentiate these drugs, and under such circumstances *Rhus* should be given the preference if it is a clear case of typhoid fever in the second or third week.

*Stramonium*.—The *Stramonium* stool is blackish and horribly offensive; the noisy delirium, has been alluded to under *Hyoscyamus*. Suppression of urine during typhoid fever is a prominent symptom.

*Sulphuric acid*.—Protracted cases, especially in children with aphthous stomatitis; stools like chopped eggs and very foetid; hæmorrhages, with rapid sinking of the vital forces; desire for stimulants. (ALLEN.) Similar to *Phosphoric acid*, but more intense.

*Veratrum viride*. — *Veratrum viride* is indicated when there is furious delirium; full, tense pulse, later becoming soft and irregular; red streak down the centre of the tongue; pneumonic complications. *Tartar emetic* may likewise be called for in dyspnœa, cyanosis, rattling of mucus in the bronchial tubes, subcrepitant râles, and œdema of the lungs.

#### DIPHTHERIA.

Diphtheria is an acute infectious, highly-contagious disease due to a specific micro-organism. While diphtheritic inflammations of mucous membranes may result from other micro-organisms—notably from the *streptococcus pyogenes*—still the term “diphtheria” should be restricted to those cases of pseudo-membranous pharyngitis and laryngitis due to the specific diphtheria germ. The other condition, described as *pseudo-diphtheria*, or *diphtheroid*, embraces those anginas complicating scarlet fever, measles, and occasionally others to the infectious diseases, or occurring primarily as “diphtheritic sore throat,” being due to infection with the streptococcus or some other organism (staphylococcus, pneumococcus).

The appearance of the membrane and the accompanying symptoms should differ greatly in these two conditions, so that a differential diagnosis might be made upon a clinical examination alone; but it must be remembered that mixed infection is quite a common occurrence, and that in such cases confusion may arise. Again, true diphtheria may present an entirely atypical exudate and the concomitant symptoms be of a mild type, while, on the other hand, a streptococcus angina may be accompanied by high fever and considerable adenopathy. For this reason it is unwise to attempt to make a diagnosis of diphtheria upon the appearance of the throat and the associated symptoms alone, never neglecting a bacteriological examination in any case presenting membranous exudate upon the tonsils.

*Membranous croup* is that form of diphtheria in which primary infection takes place in the larynx with the development of a diphtheritic membrane, which may either remain confined to the larynx or spread upward or downward, involving the pharynx and trachea secondarily.

Faucial diphtheria frequently invades the larynx secondarily, the resulting laryngeal symptoms being identical with those of a fully-developed case of croup; but the clinical picture presented by a case of primary croup differs so markedly from the manifestations of faucial diphtheria that its description, like that of pseudo-diphtheria, must be considered separately.

The *Klebs-Loeffler bacillus* is a micro-organism varying greatly in size, being broad, straight or slightly curved, and presenting a club-like extremity. It contains highly refractile, oval bodies, which take the stain more deeply than the bacillus itself, the best stain for bringing out these bodies being an acidulated solution of methylene blue, a counter-stain of aqueous Bismark brown being used to stain the body of the bacillus. This is known as Neisser's Stain and the best results are obtained in young cultures (from 6 to 12 hrs.). As the bacillus will grow readily upon Loeffler's blood-serum

at a temperature of 80° F. to 100° F., it is a simple matter to carry out this most important procedure, even in private practice. The examination of smears from the throat is neither accurate nor satisfactory. A bacillus may be found, irregular in shape and taking the stain (Loeffler's methylene blue) irregularly, but in order to be absolutely certain we must make a culture and observe its mode of growth (delicate grayish colonies) and study the early-appearing growth for the above mentioned peculiarities.

The bacillus is spread by the discharges from the mouth, throat and nose, and may persist for a long time in the throat of a patient after recovery. Infection may take place either directly from the patient, or indirectly through the medium of articles of bedding, clothing, toys, etc. It may also be spread through the agency of a third person. We should, however, remember that a child with a perfectly healthy throat is less liable to contract the disease than one with a catarrhal angina, slight superficial erosions of the mucous membrane, enlarged tonsils, naso-pharyngeal catarrh and catarrhal laryngitis.

**Pathology.**—Pathologically, a diphtheritic inflammation presents a pseudo-membrane, which is inseparably attached to the deeper layers of the mucous membrane upon which it develops, the entire mucosa having undergone a process of coagulation necrosis, accompanied by the exudation of fibrin. Such a condition is rarely met with, however, in Klebs-Loeffler diphtheria of the fauces and larynx, a croupous exudation of varying thickness, separating without leaving a lacerated surface, being the pathological process usually encountered. The true diphtheritic process, resulting in ulceration and sloughing, is more likely to take place in scarlatinal pseudo-diphtheria (septic angina), or in cases of septic diphtheria where mixed infection exists.

The membrane consists of a dense network of fibrin, containing in its meshes pus-cells, dead epithelial cells and numerous micro-organisms. The Klebs-Loeffler bacillus can



be demonstrated in the upper and outer layers of the membrane. The mucous membrane underlying and adjacent to the pseudo-membrane is found in inflammatory reaction, though rarely œdematously swollen unless pyogenic micro-organisms are plentifully admixed with the bacillus.

The lymphatic glands of the neck are markedly swollen, but do not tend to break down. The surrounding structure may present a puffy appearance. The glandular enlargement is most marked in cases complicated by invasion of the posterior nares. When the process is confined to the larynx they may not be involved at all.

Parenchymatous degeneration of the heart, kidneys and liver are the changes observed in the internal organs. A secondary broncho-pneumonia (inhalation pneumonia) is rarely absent in severe and fatal cases. Here the Klebs-Loeffler bacillus is usually found in the lungs. In sixty-two cases of broncho-pneumonia, associated with diphtheria, reported by Pearce, (*Jour. Bost. Soc. Med. Sciences*, June, 1897) the bacillus was present in fifty-two instances, being the only organism present in seventeen cases. The streptococcus pyogenes was also prominently present. The changes occurring in the nervous system, which become manifest at a somewhat later period than those observed in the other tissues, are parenchymatous degeneration of the myelin sheath of the nerves, affecting both motor and sensory fibres alike (BATTEN, *British Med. Jour.*, Nov., 1898), and at times degenerative changes in the gray matter of the cord, cerebellum and brain.

**Symptomatology.**—A typical case of pure diphtheritic infection presents the following characteristics: The child will usually complain of sore throat for a day or two, which may not attract special attention until fever, offensive breath, prostration and swelling of the glands at the angle of the jaw become apparent. An examination of the throat at this stage of the disease reveals a deposit of false membrane, usually upon one of the tonsils, associated with slight swelling of the

same and redness of the mucous membrane. This may, however, be slight, in consideration of the serious nature of the condition, and in fact the mucous membrane in some instances will appear pale rather than congested. Likewise the pain on swallowing may be so inconsiderable as to attract little or no attention. I have had children brought to my clinic in whom both tonsils were covered with membrane, and yet these children had not complained of their throats sufficiently to lead the parents to suspect the true nature of the case.

The membrane is of a grayish or yellowish-gray color, and firmly adherent to the subjacent mucous membrane; in fact, it requires more or less force to remove it, which usually results in some traumatism to the mucosa. This is a pathognomonic sign of diphtheria, and taken in conjunction with the swollen lymphatics, the offensive breath and the moderate degree of fever, a diagnosis of true diphtheria can usually be made without hesitation, which a bacteriological examination will subsequently verify. Instead of beginning as a single patch, there may be seen isolated dead-white spots of varying size upon one of the tonsils, which may remain discrete throughout the entire course, if the disease does not assume a severe type. Usually, however, they unite into one large, irregular patch, and the opposite tonsil, from being brought into contact with the affected one during deglutition, soon develops a similar membrane.

In severe cases the membrane spreads rapidly from its periphery, travelling along the margin of the soft palate, covering the uvula, which becomes elongated and swollen, and finally invading the opposite half arch and coalescing with the membranous deposit of the other tonsil. It also spreads posteriorly to the pharynx, whence it may invade the posterior nares or the larynx. When membrane develops at this rapid rate it sometimes appears simply to run over the mucosa as a delicate fibrinous exudate, the epithelium beneath it remaining intact for some time.

In a steadily progressing case the above distribution of the membrane will have been completed in about three to four days from the time of onset. At this time the membrane can be studied in various stages of development. At the site of origin it will be found to have attained considerable thickness, being of a brownish or dirty grayish color, with a well-defined outline and areola, and a thick, partly detached border, while in another direction it fades out into a thin, grayish film, which is invading new territory. This film likewise thickens and assumes the same color as the other portion of the membrane, which now shows a tendency to become loose.

By the fifth or sixth day the process has reached its acme, and in the course of three or four days the membrane separates spontaneously, providing the patient has not succumbed to the disease or has not had antitoxin. (In the cases receiving antitoxin early, the course is materially shortened.) A red areola of reactionary inflammation is seen about its border, and it gradually loosens and comes away in pieces, leaving behind a reddened, slightly swollen and readily bleeding mucous membrane. Coincident with these changes the constitutional symptoms rapidly improve, and the patient is on the road to convalescence.

The symptoms accompanying the diphtheritic process are those of a most grave toxæmia. As pronounced symptoms may be delayed until the disease is far advanced, they are seldom of diagnostic value. The child complains of lassitude, anorexia and sore throat; repeated chilly sensations and headache may also be present. Fever is usually not high in the beginning, and may remain at a comparatively low point throughout the entire course, fluctuating between  $101^{\circ}$  to  $103^{\circ}$  F. An abrupt onset, however, with high fever, headache, severe pain in the throat and considerable swelling of the tonsils, may take place. In such cases the early symptoms may be due to an admixture of the streptococcus. This, however, is not necessarily the case, for diphtheria is most irregular in its clinical manifestations. There are cases in which even in the presence of considerable

membrane constitutional symptoms are entirely wanting. Again, the most serious symptoms, even sloughing of the soft tissues and suppuration of the lymphatic glands may result from the diphtheria bacillus alone without the intervention of a secondary infection.

The lymphatic glands at the angle of the jaw are involved early in a typical case, but the adenopathy may be so slight, as to escape notice. Absence of adenopathy is by no means evidence against the presence of diphtheria.

The pulse becomes rapid and weak during the later stages, the heart being affected to a marked degree by the toxins of diphtheria. Sudden death may take place from cardiac paralysis during the height of the disease, or it may not occur until the child is convalescing, following upon some incautious physical exertion. The myocardium, as well as the innervation of the heart, is affected by the toxin. In some cases the pulse becomes slow and irregular; this is probably a sign of myocarditis.

The tongue is coated from the beginning and the breath characteristically offensive. The bowels are generally constipated. Albuminuria is found in many cases; it usually clears up promptly, simply indicating acute degeneration of the kidneys as a result of the elimination of toxins.

**Diphtheritic paralysis** occurs more frequently in adults than in young children, being seen seldom under two years. The severity of the case does not necessarily indicate the amount of paralysis which is to be expected, for cases with but a small amount of membrane may be followed by considerable paralysis, and *vice versa*. The clinical picture is that of a multiple neuritis, the pathological changes in the nerves having been described above. Symptoms may occur while the membrane is still present, but this is unusual. In the majority of cases they do not occur until two or three weeks after recovery. Paralysis of the soft palate is the first symptom noticed, manifesting itself by nasal voice, regurgitation of food through the nares, and difficulty in swallowing.

The eye-muscles are frequently affected early, and loss of accommodation, strabismus and ptosis are the disturbances encountered here. When the extremities take part in the paralysis the patient will complain of muscular weakness, with tingling and numbness, gradually increasing in severity until he is perhaps unable to walk or use his arms, although complete paralysis is rare. When the extremities become involved the paralysis is symmetrical. Sensation is markedly impaired and the knee-jerk lost, even at times without the existence of paralysis. The prognosis as to ultimate recovery is good, although the course is variable, some cases continuing for several months before improvement sets in. Death may result from paralysis of the respiratory muscles.

*Extension of the membrane to the nose* is indicated by nasal obstruction with an acrid, offensive, muco-purulent discharge and increased swelling of the lymphatic glands at the angle of the jaw, together with involvement of the submaxillary glands. Epistaxis occurring during diphtheria is always a suspicious symptom. Owing to the large absorbing surface brought in contact with the toxins, constitutional symptoms are markedly aggravated, and prostration becomes extreme. *Primary nasal diphtheria* is, as a rule, not nearly as grave a condition as the secondary form, although such a case may infect another child with a faucial diphtheria of the usual severity. (For a full discussion of nasal diphtheria see *Pseudo-membranous Rhinitis*, pages 549 and 550.)

*Extension into the larynx* is indicated by progressively increasing dyspnoea, cyanosis, and a croupy cough. The process may result in complete stenosis of the larynx, with death from suffocation.

*Septic diphtheria* is characterized by the addition of sepsis to the diphtheritic condition. It was formerly supposed that this form of diphtheria is invariably due to mixed infection with the streptococcus, but it is now known that even the most virulent cases with all the outward signs of a septic infection may be due to the Klebs-Loeffler bacillus alone. The

throat assumes a dirty grayish color, or even blackish where blood extravasation into the false membrane has taken place, and a cadaverous stench emanates from the mouth. A tenacious brownish mucus covers the tongue and lips, and an acrid discharge runs from the nostrils. The lips are dry, swollen and cracked, and may be covered with patches of false membrane. Swelling of the lymphatics at the angle of the jaw is pronounced, and is accompanied by infiltration of the cellular tissue of the neck. The pulse is rapid and feeble, the extremities become cold, and prostration is profound. The temperature fluctuates greatly, and in a given case may range from subnormal to a hyperpyrexia. Septic cases are usually rapidly fatal, succumbing to the toxæmia more often than to laryngeal involvement.

**Laryngeal Diphtheria** or **Membranous Croup** is a primary infection of the larynx characterized by the formation of a false membrane (croupous exudate) upon the laryngeal mucous membrane. The false membrane may remain confined to the larynx, or extend down into the trachea and up into the pharynx. Often it is accompanied by a scanty tonsillar exudation. Laryngeal diphtheria presents few of the characteristic symptoms of faucial diphtheria for a number of reasons. In the first place, the bacillus causing croup is usually less virulent than that found in faucial diphtheria. Again, owing to the feeble absorptive power of the mucous membrane lining the larynx, glandular enlargement does not take place, and as constitutional symptoms are delayed for the same reason, they are not frequently observed, owing either to the rapidly fatal course of the disease or to a checking of the process before symptoms have had time to develop. Goodno states that the fatal cases of primary pseudo-membranous laryngitis observed by him which were subjected to tracheotomy, and lived long enough to develop constitutional symptoms, died as diphtheria patients die.

The onset is insidious, with moderate fever, croupy cough, and hoarseness. During the first few days symptoms are

slight and only point to a catarrhal laryngitis, nocturnal aggravations frequently occurring from spasm of the vocal cords. When, however, an exudate is seen upon the tonsils, or down in the pharynx, we are justified in suspecting the diphtheritic nature of the case. At the end of from three to four days laryngeal obstruction has become the chief feature in the case. The voice is hoarse or entirely lost; during inspiration a harsh, tubular sound (stridulous respiration) is heard, and the act is accompanied by retraction of the supraclavicular and intercostal spaces and the lower border of the thorax. Recession of the epigastric region during inspiration is a sign by which we can most satisfactorily gauge the degree of laryngeal obstruction in young children. The child usually sits erect, and every effort at inspiration is laboriously performed, all of the accessory respiratory muscles being thrown into action. With progressing stenosis the body surface becomes cold and cyanotic, and the child becomes drowsy and later comatose, dying from asphyxia. Death may result in a few days from the time of onset, although the course is usually somewhat longer. With the intervention of surgical measures (intubation and tracheotomy) the case presents a less unfavorable prognosis.

**Pseudo-Diphtheria**, or **Diphtheroid**, differs from true bacillary diphtheria both etiologically and symptomatically. Such a diphtheritic process may develop independently or complicate scarlatina, measles, etc. As a complication of scarlatina it appears, however, more frequently and more virulently than in any other form. In this disease an angina of almost any grade of severity seems possible, the virus of scarlatina exerting direct and specific influence upon the throat, and permitting of the development of the gravest forms of diphtheritic inflammations.

The *streptococcus pyogenes* is the germ most frequently found in pseudo-diphtheria, as was first demonstrated by Prudden. Although other micrococci, notably the *staphylococcus aureus* and *albus*, are sometimes found alone in these



cases, or in association with the streptococcus, still they play a less important rôle than the latter, which is capable of producing the most destructive manifestations. The work of Filatow (*Vorlesungen ii. Infections-Krankheiten im Kindesalter*, 1897) fully confirms these observations, and he is led to the belief that all "scarlatinal-diphtherias" are streptococcus anginas; furthermore, pseudo-diphtheritic streptococcus angina may be encountered as an independent disease, occurring without scarlet fever. Holt states that from 25 to 35 per cent. of cases formerly sent to hospitals with a clinical diagnosis of diphtheria were really cases of pseudo-diphtheria.

Vierordt (*Berliner Klin. Wochenschr.*, 1897) found in a series of diphtheroid anginas both streptococci and staphylococci, and in one case a diplococcus. The Klebs-Loeffler bacillus was present in none of these cases. The membrane did not extend to the nose or pharynx, but in the greater number it passed beyond the tonsils in the direction of the soft palate. Bouloché (*Les Angines à Fausses Membranes*) divides the various pseudo-diphtheritic anginas into the following classes: *streptococcus angina*, *staphylococcus angina* and *pneumococcus angina*. He considers them non-contagious and usually mild in their course. I have encountered the pneumococcus in a few of my cases.

Although these bacteria are found as contaminations in most cases of diphtheria, still they do not modify the course of the disease unless present in large numbers, and even then it is only the streptococcus which materially alters the nature of the case, the bacillus-streptococcus combination being the most unfavorable form of infection, producing a septic diphtheria.

The *clinical course* of pseudo-diphtheria is quite different from that of bacillary diphtheria. In cases of mixed infection a clinical differentiation becomes difficult or impossible. In pseudo-diphtheria there is pronounced inflammation of the pharynx and tonsils, with redness, swelling and pain. It be-

gins abruptly, with high fever, lassitude and headache. Soon small, white or yellowish patches are seen to develop upon the tonsils; they become darker in color and may coalesce, but seldom spread beyond the tonsils. The membrane is more friable than that of true diphtheria, and can usually be detached without much difficulty.

Swelling of the lymphatics seldom takes place. Such cases run a comparatively short course—from four to five days—and although constitutional symptoms are severe during the height of the disease, the throat symptoms being particularly distressing, still they are never dangerous in character, and sequelæ are rare. Of course, albuminuria and even an endocarditis may complicate such a condition (see Acute Tonsillitis, p. 537). Paralysis never follows pseudo-diphtheria, nor is extension to the larynx to be feared, although in the severer cases which complicate scarlatina extension to the nose and Eustachian tubes frequently takes place.

*Scarlatinal pseudo-diphtheria* may become a very serious condition. Beside the extension of the membrane just alluded to, sloughing and ulceration may occur, with general septic infection, and cellulitis of the neck and suppuration of the lymphatics. Such cases present a high mortality rate, being equalled in virulence by septic diphtheria only, from which they cannot be separated except by a bacteriological examination. The membrane develops during the height of the fever in the majority of cases, but it may be seen before the eruption appears. A diphtheritic sore throat developing after the fever has abated, or during convalescence, is more likely of bacillary origin than one developed at the height of the disease.

**Prognosis.**—In estimating the prognosis in a given case of diphtheria several factors must be taken into consideration. In the first place, we must exclude pseudo-diphtheria, which in its primary form offers a good prognosis; in its secondary form (scarlatinal) the prognosis is less favorable and it assumes more the type of a septic infection.

The age is of importance, as diphtheria is uniformly more fatal in infants than in older children. Adults present the best chances, but they are more subject to paralytic sequelæ.

The character of the epidemic is of importance, as is also a knowledge of the source of infection. But this is not always reliable, for a most virulent diphtheria may originate from an apparently mild diphtheritic sore throat, and *vice versa*.

The appearance and distribution of the membrane offer valuable suggestions for the prognosis, but here again errors are liable to occur. Extensive membranous deposit may exist with but slight constitutional disturbances, and scanty membrane may be accompanied by grave toxæmia. Neither can we foretell if laryngeal involvement, with rapidly developing stenosis, will occur.

The time at which treatment was begun and the patient's general condition, therefore, offer the safest guides in determining his chances for recovery. So long as the pulse remains good and prostration is not pronounced the case should not be despaired of. Nasal and laryngeal diphtheria are about equally grave, although the nasal type is somewhat slower in its course. Septic cases are practically hopeless. Other unfavorable symptoms are epistaxis and hæmorrhages into the subcutaneous tissues; nephritis; marked prostration and cardiac weakness; cervical cellulitis.

In *croup* the prognosis is more favorable than in secondary laryngeal diphtheria, owing to the absence of septic symptoms.

During convalescence there is danger of paralysis of the heart. This may appear as a progressively increasing heart weakness, or occur suddenly upon some physical exertion. The child is seized with epigastric pain and nausea; there is dyspnœa; cyanosis; small, irregular pulse and collapse. If the first attack does not prove fatal there is usually a recurrence with a fatal issue.

Broncho-pneumonia occurring with diphtheria is very unfavorable; when complicating croup the case is practically

hopeless, as intubation or tracheotomy is of no avail in such cases.

**Diagnosis.**—There is only one safe means of escaping the error of allowing a case of diphtheria to gain headway unrecognized until so far advanced as to be self-apparent, and that is to examine the throat of every child presented for treatment in an acute condition, as a matter of routine. The importance of such practice is realized only when we recall how trivial the throat symptoms may be in the beginning of diphtheria, particularly in a child not able to express itself or comprehend its sufferings properly.

The differential diagnosis rests mainly between *pseudo-diphtheria* and *folliculous tonsillitis*. *Pseudo-diphtheria* is abrupt in onset; lymphatic swelling is absent in primary cases; fever is high, and the throat is markedly reddened and swollen, and there is considerable pain on swallowing; the exudate is purely fibrinous, rarely croupous, and it does not tend to spread beyond the tonsils. Secondary cases occur during the febrile period of scarlet fever. Paralysis never follows, and although septic symptoms may be present the specific toxic symptoms of diphtheria are absent. The membrane is thinner, can be removed without bleeding, and is usually of a yellowish color, later becoming dirty.

In *folliculous tonsillitis* both tonsils are uniformly swollen and covered with small, round, white spots, which are not adherent to the mucous membrane, but consist of plugs of exudation filling up the lacunæ of the tonsils, from which they can be readily expressed and wiped off.

*Membranous croup* is to be differentiated from *acute catarrhal laryngitis*. (See p. 257.)

Lastly, it may be said that no diagnosis is complete without a bacteriological examination, for a case which may appear clinically of minor importance may harbor germs of a most virulent nature. The differentiation of pseudo-diphtheria from true diphtheria becomes also of the greatest importance in the matter of isolation, particularly in avoiding the deten-

tion of patients suffering from the former disease in isolating wards harboring true diphtheria.

**Treatment.**—Isolation and sick-room hygiene are to be carried out on the same plan as recommended under *Scarlatina*, page 650. Children who have been exposed to diphtheria should have their throats examined several times daily, and be instructed to use a gargle of *Permanganate of Potash* (1 to 1,000) three or four times daily. Royer (*Therapeutic Gazette*, April, 1905) insists that the general practitioner does not resort with sufficient frequency to *immunizing* doses of anti-toxin. He commonly sees at the Philadelphia Municipal Hospital a patient admitted seriously ill with diphtheria, and in the course of a few days a second or third patient from the same house. For a child that has come in direct contact with a diphtheria case he recommends one thousand units. In my own practice I carry out this principle whenever possible.

The *diet* must be of a most concentrated and nutritious form, and stimulation is of the greatest importance as soon as the toxic influence of the diphtheria virus upon the heart and nervous system becomes apparent. A teaspoonful of whisky well diluted with water or milk, and administered every two to three hours, suffices for the average case; but where there is much prostration and failing heart the quantity must be increased accordingly. Absolute rest is to be enjoined during convalescence as well as during the disease in all cases showing cardiac weakness, in order to avert a possible sudden death.

As to *local treatment*, it can be positively stated that all measures in any way giving the patient pain or discomfort and requiring physical restraint, or resulting in injury to the mucous membrane of the throat, will do nothing excepting harm. In infants a spray of *Permanganate of Potash* (1 to 1,000) given, by means of an atomizer, every two or three hours, and in older children a gargle similarly employed, has yielded the best results in my hands. Should the

child be too weak to gargle, a teaspoonful of the solution may be given internally every two hours. Alcohol diluted with four or five parts of water is also an excellent gargle, but not as active as the *Permanganate*.

In *nasal diphtheria* our aim should be to keep the nasal chambers as open and free from secretion as possible. A douche of *Permanganate*, 1 to 2,000, or a warm normal saline solution should be given about three times daily, as directed on page 19, but the child should be held in the recumbent position with its head turned to one side, lying on a Kelly pad.

In *laryngeal diphtheria* an emetic will give temporary relief when suffocation becomes imminent, but intubation or tracheotomy should not be put off to so late a period. Although still a matter of dispute, intubation is the preferable procedure in the majority of cases. It should always be attempted first, and, in the event of not offering the most desirable results, tracheotomy may be resorted to as a *dernier ressort*.

**Intubation** consists in the introduction of a hard rubber tube into the trachea by means of an especially constructed instrument invented by O'Dwyer. In construction it is practically nothing more than a handle, to the end of which, at a right angle, the intubation tube is temporarily fastened by being slipped over an obturator. From this it is released at the proper moment by means of a hook-like arrangement that is pressed down over the collar of the tube and thus pushes it loose from the obturator (Fig. 60). Extubation is performed by means of a long, curved, forcep-like instrument with a small beak, as shown in the illustration. The beak is inserted into the opening of the tube, the blades are separated until the tube clings to the same, and the tube is then withdrawn from the larynx.

The child may be intubated in the erect or in the recumbent posture. Personally, I find the recumbent posture preferable, and it is employed in the majority of the hospitals that I have

visited. The child is easier to control in this position, and, furthermore, the palate tends to fall upward and away from the pharynx, thus facilitating the introduction of an instrument into the larynx.

The child is prepared by being wrapped in a sheet with the arms pinned down to the sides, and the nurse is instructed to hold the legs and trunk, while an assistant should be at hand to control the head and keep the



FIG. 60. O'DWYER'S SET OF INSTRUMENTS FOR INTUBATING THE LARYNX.

mouth-gag in place. A table covered with several thicknesses of blankets and a sheet should be used, and not a couch or bed. The mouth-gag is then inserted into the left side of the mouth, well back between the molar teeth. The proper tube having been selected, according to the age of the child, and threaded with a loop of heavy linen thread, it is now slipped firmly over the obturator of the introducer. The latter is lightly held in the right hand, with the thumb upon the knob



of the sliding arrangement which releases the tube. The left index-finger is passed quickly into the child's pharynx, keeping to the right side of the mouth, as shown in Fig. 61, and the epiglottis is hooked up and held out of the way so that the tube may be guided along the middle line, over the base of the tongue, and then directly downward and slightly forward into the chink of the glottis. In order to give the proper direction to the tube the handle of the instrument must be

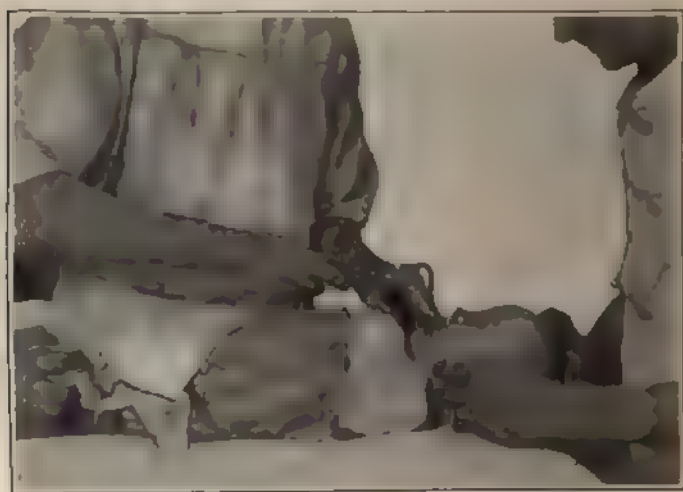


FIG. 61.—CHILD IN PROPER POSITION FOR INTUBATION. INDEX FINGER OF LEFT HAND IN MOUTH, FEELING FOR THE EPIGLOTTIS. RIGHT HAND HOLDING INTRODUCER WITH TUBE READY FOR INTRODUCTION.

well elevated as soon as the tip of the tube reaches the glottis. When the tube has entered the glottis it should be released (by means of the slide on the introducer, which is pushed forward with thumb), and then buried to its full length into the larynx by a gentle push with the left index finger upon the collar of the tube. No force is necessary, and undue pressure or rough manipulation of the larynx is strictly to be avoided.

Should the first attempt fail to enter the tube promptly

within the glottis, the finger should be removed from the pharynx and the child allowed to get its breath before making another attempt.

The commonest mistake is to pass the tube into the œsophagus instead of into the larynx. This results from not elevating the handle of the introducer sufficiently and failing to direct the tube sufficiently forward; also from getting away from the middle line and not keeping closely to the base of the tongue. The most reliable landmark by which to find the larynx is the arytenoid cartilage. Even when the mucous membrane is swollen and false membrane is present this knob-like projection can readily be distinguished by the index finger. From this we may pass a little inward and forward in order to find the epiglottis, which must be lifted up before the tube can enter the larynx.

If we have been successful in our attempt, a remarkable change soon passes over the child. There will first occur a coughing paroxysm, more or less severe, accompanied by a characteristic whistling sound which tells us that the tube is in the larynx. The cough results from the mechanical irritation of the larynx, but this is beneficial, as it usually effects the removal of a large amount of tenacious mucus. The normal color is restored to the features, and the breathing, which was rapid and labored, becomes slow and tranquil. Usually the child soon goes off into a refreshing sleep. As soon as we are convinced that the tube is in place and the dyspnœa is relieved, we should cut the string that has been attached to the tube (mainly for the purpose of pulling it out of the œsophagus in an unsuccessful attempt) and draw it out of the eyelet in the collar of the tube with the left index finger in position upon the collar in order to avoid an accidental extubation.

At the end of the fifth day the tube should be removed. The child is held exactly as for intubation and, with the left index finger upon the collar of the tube, the beak of the extubating instrument is guided into the opening of the tube,

its jaws are opened until they will not slip from the opening, and thus gripping the tube it is lifted from the larynx. It can then be easily removed from the pharynx with a quick sweep of the index finger.

We must always be ready to re-intubate promptly, however, as there may still be sufficient stenosis to render the wearing of the tube necessary, or a spasm of the laryngeal muscles may follow the removal of the tube, calling for prompt action. In rare cases the intrinsic muscles of the larynx become paretic from the pressure of the tube, or a cicatricial atresia of the larynx may follow upon pressure-ulceration. Fortunately, the beneficial results from skilfully performed intubation so far outshadow its evil results that it has attained to a position of universal praise as a life-saver.

One of the disadvantages of intubation in private practice is the possibility that the child may cough up the tube and suffocate before the physician can return to the case. It is well to remain with every case, or at least close by, for several hours after intubating, and if the child shows a tendency to cough up the tube it should be removed, whenever possible, to a hospital where skilled residents are in charge. Death may result under somewhat similar circumstances, even after tracheotomy; the child may pull the tube out or it may become clogged with membrane. The eminent pædiatrist, Prof. Caillé once remarked, we can do no more than perform our duty under these circumstances, and if we have done the best we know how, results may come as they will.

The membrane may extend down too low to enable the intubation tube to relieve the dyspnœa, or we may push the membrane down ahead of the tube and thus cause suffocation. In the latter instance a violent coughing fit will sometimes expel both the tube and the membrane. These difficulties are mainly encountered in the cases we see late, and I have been forced to do tracheotomy several times under such circumstances. But we should not wait until the child is *suffocating* before intubating. As soon as the breathing becomes

laborious and the epigastric region is plainly drawn in during inspiration it is our duty to spare the child not only from the suffering, but also from the exhaustion that comes with these undue respiratory efforts.

*Nasal intubation* as recommended by Northrup (New York) merits serious consideration. It is indicated in complete obstruction of the nose by membrane, in young children and infants, when this obstruction materially interferes with the child's respiration. An old-fashioned English (stiff) catheter of the proper calibre is carefully worked through the naris until the pharynx is reached; it is then cut off, allowing about an inch to protrude. Tubing one side may give sufficient relief in the case. Dr. H. M. Gay has reported a case which I saw with him and treated in this manner, the infant making a good recovery (*Tran. Hom. Med. Soc., Penna., 1904*).

In all forms of diphtheria, but especially in croup, it is essential to keep the air of the room moist and at a temperature of about 70° F., if this be practicable. The air must, at the same time, be kept as rich in oxygen as possible. The spraying about the sick-room of hydrogen dioxid, or the slaking of lime, is an excellent means of purifying the air.

It is by no means easy to say just which are our most important remedies in diphtheria, as no attempts at differentiation have been made in the past between pseudo- and true diphtheria, and the errors which so frequently beset the diagnosis of membranous croup render it difficult to estimate the exact value of the treatment employed.

The *Mercuries*, especially the *Cyanide*, the *Bichloride* and *Red iodide*; the *Bichromate* and *Permanganate of Potash*, the *Chloride of Lime*, *Lachesis*, *Arsenic* and *Arum triphyllum* are most closely related to the bacillary variety. *Merc. cyan.* and *Kali bich.*, especially when there is extension to the larynx; *Arum triph.* in the nasal variety, and *Arsenic*, *Lach.*, *Rhus tox.* and the *Chloride of Lime* in septic cases.

The high-grade inflammatory symptoms of pseudo-diphtheria call for remedies like *Apis*, *Belladonna*, *Ailanthus*, *Phytolacca* and *Rhus tox.*

Mild diphtheritic anginas yield promptly to the *Mercuries*, *Bell.* and *Apis*. The *Red iodide* is supposed to be indicated when the left tonsil is affected, and the *Yellow iodide* when the right side is involved. Personally, I do not pay any attention to the side affected, and always give the *Red iodide* the preference over the *Yellow*. In *Belladonna* there is considerable fever and headache, the throat is dry and glistening red, and there is pain in swallowing and a sense of constriction. When these symptoms are present with considerable exudate I alternate *Belladonna* with the *Red Iodide of Mercury*. *Apis* presents more of an œdematous condition, the swelling being paler in color and the pains of a stinging character, worse on swallowing.

The most efficient remedies in croup are *Bromine*, *Iodium*, *Kali bichromicum*, *Liquor calcis chlorinata* and *Hepar*. The symptoms of *Spongia* are more purely catarrhal and spasmodic than croupous. *Bromine* has given me good results in a few cases, but since the introduction of antitoxin the mortality of croup has been so much reduced that we have no right to rely exclusively upon a remedy. Dunham's experience (*The Science of Therapeutics*) with Boënnighausen's method of prescribing *Aconite*, *Hepar* and *Spongia* in rotation seems to have been of the happiest kind. Neidhard obtained good results from his *Liq. calcis chlorinata* in croup as well as in faucial diphtheria, but he frequently alternated with *Potassium bichromate* in the former condition.

The routine treatment advised by Heysinger (*Journ. of Ophthal., Otol. and Laryngol.*, January, 1892) is of great value in all cases of pseudo-diphtheria and in diphtheria with septic symptoms. He administers a teaspoonful of a solution of *Permanganate of potash*, one grain dissolved in two and one-half to three ounces of water every one to two hours, according to circumstances, in alternation with a teaspoonful of *Belladonna*, five minims of the tincture in three ounces of water. The *Belladonna* relieves the fever and hyperæmia; but with toxæmia it is of no value, and a remedy must be

chosen symptomatically (*Merc. cyanat.*, *Arsenic*, *Lachesis*, *Rhus toxicodendron* and others).

The symptoms upon which the remedies applicable to diphtheria and croup have been prescribed most frequently are the following:

*Acetic acid*.—Croup, attended by bright redness of the face. From five to ten drops of acetic acid in a half tumblerful of water with some sugar; a teaspoonful every two to three hours (C. G. R.).

*Ailanthus*.—Scarlatinal diphtheria (diphtheroid) with livid and swollen throat. Deep ulcers on tonsils; the patient gradually sinks into a stupor.

*Ammon. carb.*.—Nasal obstruction and carbonization of the blood; extreme prostration.

*Apis mel.*.—Œdematous swelling of the fauces, cellular tissue of neck and of the glottis. Burning and stinging pains in throat; albuminuria.

*Arsen.*.—In the later stages, especially in toxic cases with marked cardiac weakness; albuminuria; irregular fever; extreme restlessness.

*Arum triph.*.—Acrid discharge from the nose excoriating the upper lip; an acrid fluid oozes from the mouth which causes the lips to become sore and swollen. The child constantly picks at the lips and nose, keeping them in a bleeding condition. There is burning pain in the throat, and the breath is very offensive. The membrane spreads up into the nares.

*Bell.*.—Early, especially in cases beginning abruptly with pronounced throat symptoms and fever.

*Bromium*.—Croup. Best suited to fair, chubby children, and in cases with little or no fever. *Iodium* is recommended in brunettes and in the presence of fever.

*Calc. chlor.*.—The *liquor calcis chlorinata* was first recommended by Dr. Neidhard (*Diphtheria, Its Nature and Homœopathic Treatment*, 1867) in diphtheria and croup, and it is still a favorite remedy with many practitioners. He em-

ployed five to fifteen drops of the *Liquor* in half a tumbler of water, giving a teaspoonful every fifteen minutes in urgent cases, or only at intervals of several hours in more favorable ones. His success was apparently most gratifying.

*Hepar.*—Croup. The cough is hard and metallic, with a loose edge ; although the child may expectorate, the obstruction is not relieved. Croup developing after exposure to cold wind.

*Kali bichr.*—Croup. Tough, stringy discharge from throat, with hoarseness and croupy cough. Also nasal diphtheria with a similar discharge from nose ; extension up into the Eustachian tube. The best remedy to control the excessive secretion in croup cases even after antitoxin has been administered.

*Lachesis.*—The symptoms are intense, although the throat lesion may be apparently slight (toxic cases). The membrane is grayish or becomes black, (hæmorrhagic) and is surrounded by a purplish areola ; the throat is purple, not bright red as in *Apis* (Allen) ; the cellular tissue of the neck is infiltrated and the skin presents a livid hue. Hyperæsthesia about the throat is a most characteristic symptom, and the patient must have all garments as loose as possible in this region.

According to Allen, in all cases requiring *Lachesis* there is usually sharp pain shooting from the throat up into the ears. Cardiac symptoms may also be prominent, the patient being unable to get his breath unless propped up in bed, etc.

*Merc. cyanatus.*—Adynamic cases with abundant membrane displaying a tendency to travel down into the larynx. "Adynamic fever and collapse already in the commencement." (VON VILLERS.) According to Allen it is also of service in nasal cases with profuse debilitating sweat from the slightest exertion. The *Cyanide of Mercury* is undoubtedly a truly homœopathic remedy to toxic diphtheria, producing the extreme adynamia observed in these cases as well as gangrene of the velum palati and fauces (BECK). Of late the old school has been making use of it, a series of eighty-one



cases of diphtheria with but one death being reported by Luddeckens-Leignitz (*Aerztliche Rundschau*, 1896, No. vi). He also uses it in scarlet fever, whether complicated by membranous angina or not. His usual dose was a teaspoonful of a 1 to 10,000 solution every hour (fourth decimal dilution).

*Merc. jod. ruber.*—Membrane begins on the tonsils; painful swelling of lymphatics; the tonsils are swollen and the palate elongated; the patches are irregular in outline and of a dirty yellowish color; tongue heavily coated and flabby; offensive breath. The *Yellow iodide*, it is claimed, affects the right side and partakes more of the general characteristics of *Mercury*, while the *Red iodide* displays more of the action of *Iodine*. I do not believe, however, that there is sufficient clinical experience to give the proper authority to this distinction.

*Phytolacca.*—Much pain and swelling in the throat. The mucous membrane is of a bluish-red color and is covered with grayish ulcers.

*Rhus tox.*—The throat appears as if varnished (glistening), and of a dusky red color. Swelling of the lymphatics and cellular tissue of the neck is marked. There is great restlessness and prostration, with aching in every joint. Septic cases.

**Post-diphtheritic Paralysis.**—The most useful remedies are *Gelsemium*, *Causticum*, *Phosphorus*, *Cocculus* and *Nuxvomica*. *Strychnine* is extensively used by the old school, but many of its best authorities are in doubt as to any specific influence exerted by it over the condition, relying more on a general tonic treatment and galvanism. Stiegele (*Allg. Hom. Zeitung*, Dec., 1901) reports a case in which *Phosphorus*, 5th dilution, was prescribed with rapid improvement, undoubtedly due to the drug's action, on the following symptoms: Cardiac weakness with spells of palpitation; marked debility of the extremities; uncertain gait, and formication in the hands and feet. *Causticum* is most useful in laryngeal and ocular paralysis. Cardiac weakness with blueness of the lips and ptosis call for *Gelsemium*.

**Serum Therapy.**—The antidotal treatment of diphtheria, by means of the hypodermatic injection of the blood-serum of horses, previously immunized to the toxins of diphtheria by being subjected to progressively increasing doses of the same, has furnished us with the most valuable means at our command for the cure of this much dreaded disease. Not only is the serum, popularly known as *diphtheria antitoxin serum*, capable of antidoting the systemic disturbances belonging to diphtheria, but it also exerts a specific influence upon the local manifestations. Furthermore, it has been conclusively demonstrated that an artificial immunity against the disease can be obtained from a comparatively small dose. This, however, is of a transient nature, only lasting for a period of a few weeks, but it nevertheless indicates the strong antagonism which exists between the serum and the toxin. The overwhelming evidence in favor of this form of treatment in diphtheria, based on statistics coming from both Europe and America, and from private practice as well as from large hospitals for contagious diseases, should expel all doubt as to its efficacy. When we consider that the clinical manifestations of diphtheria are purely of toxic origin, requiring antidotal treatment as well as any other case of poisoning, be it a snake bite, arsenic- or opium-poisoning, we should gladly take advantage of such a potent remedy, and by using it in conjunction with judicious local measures and remedies chosen on indications requiring special consideration, we may hope to still further lower the death-rate so materially changed within the last decade.

The sphere and scope of antitoxin must not be superficially considered, for it has both its limitations and characteristic indications, as well as any other remedial agent of positive value. Again, the limit of its action is to antidote the toxins circulating in the blood and check the local process; it has no curative effect beyond this, which is simply the creation of an artificial immunity. Upon the parenchymatous changes

in the heart, kidneys and other internal organs, and the specific changes in the nervous system, it has not the slightest influence. Consequently its efficacy becomes less and less positive as the disease is allowed to progress without efforts to check it, and the occurrence of diphtheritic paralysis cannot be averted in a system which has already been saturated with the toxins, even if antitoxin be used late in such a case with otherwise favorable results. In pseudo-diphtheria and in septic cases it is useless. The use of antitoxin in such cases has given it many a black eye, if I may use such a term. If a serum is at all to be used here, it must be one capable of neutralizing streptococcus toxins. A slight admixture of streptococci cannot be said to contraindicate the employment of antitoxin, as the bacillus produces the most important disturbances in these cases; but if there be considerable angina and adenitis, fever, headache and other symptoms of a similar nature, the best results are obtained by using the *Belladonna* and *Permanganate of potash* combination in conjunction with the antitoxin.

A perfect case for antitoxin is one in which the bacillus is found in practically pure culture in the throat and the symptoms correspond to the description of a typical case of uncomplicated diphtheria as depicted above. Membranous croup of purely bacillary origin is also most positively benefitted by it. As pseudo-membranous laryngitis only exceptionally results from other than diphtheritic infection, the exceptions being those rare cases accompanying malignant scarlatina, it possesses in antitoxin a remedy for which we should indeed be thankful.

According to the report of the collective investigation of the American Pædiatric Society (*New York Medical Record*, May 15, 1897), the mortality among cases of laryngeal diphtheria operated upon was reduced to 27.24 per cent., early statistics of intubation in pre-antitoxin days showing only 27 per cent. recovery. The number of cases requiring operation was also greatly reduced, being 39 per cent. with the use of

antitoxin, and about 90 per cent. without it. In a former report it was shown that the average mortality from faucial diphtheria in private practice was about 12 per cent., but among the cases which received antitoxin within the first three days it was only 7.3 per cent. Prof. Goodno, who was one of the first in our school to champion the serum therapy of diphtheria, reported two hundred and seventeen cases of diphtheria seen in private practice (mostly in consultation) with nine deaths, a mortality of about 4 per cent. (*Hahne-mannian Monthly*, June, 1901). Baginsky treated eighty-two consecutive cases with antitoxin at the Friedrich's Hospital in Berlin, with a mortality of 12.2 per cent. Immediately following this series, one hundred and three cases were treated without antitoxin, and the mortality rose to 53.4 per cent. After this, antitoxin was resumed, and out of one hundred and twenty-four cases only 11.3 per cent. died. Clubbe (*British Medical Journal*, Oct., 1897) reported a parallel series of three hundred cases of diphtheria, treated with and without antitoxin, at the Sydney Children's Hospital. The diagnosis was confirmed bacteriologically in all cases. Of those treated without the serum, 52.7 per cent. died; one hundred and ninety-nine required tracheotomy, with a mortality of 67.8 per cent. The mortality was reduced to 20 per cent. by the employment of serum injections, and among this series only one hundred and twenty-nine required tracheotomy, with a mortality of 37.9 per cent. Since antitoxin has been used more heroically even better results are obtained. Thus, in Boston, McCollom (City Hospital, South Dept.) gradually brought his mortality rate down to about 11.5 per cent. in 1903, from 14.5 per cent. in 1895-'96,—the early period of serum therapy. In intubation cases it fell from 64.5 per cent. in 1896 to 26.6 per cent. in 1903. Statistics could be cited *ad infinitum*, but as they all practically indicate the same beneficial results from the antitoxic treatment the foregoing will suffice.

Accurate rules for dosage cannot be laid down for all cases,

nevertheless there are certain rules of procedure that apply to the average case. In the last few years the views concerning the dosage of antitoxin have changed considerably, especially since McCollom, has so clearly demonstrated the harmlessness of large doses and their advantage over the smaller dose.

The following doses are in use at the Municipal Hospital (Philadelphia). Purely tonsillar exudate (single), 2,500 units ; the same, double, 5,000 units. Tonsillar exudate with involvement of pillars and uvula and larynx, 7,500 to 10,000 units ; nasal and any other part involved, 7,500 to 10,000 units ; laryngeal, 7,500 to 10,000 units.

Repeat the dose in each case in from twelve to twenty-four hours, depending upon severity of case and signs of improvement (ROYER, *loc. cit.*).

I doubt the necessity for such doses in private practice, especially when we see the case early. In mild cases, seen early, 2,000 units usually suffice. In the severe cases we should begin with 4,000 units and repeat the injection at the end of twelve hours if the progress of the disease has not been arrested. If the patient is worse I recommend double the initial dose (8,000 units) to be given at this time. If improvement sets in, but the membrane does not promptly come away, the injection should be repeated at the end of twenty-four to forty-eight hours.

In laryngeal cases it is best to give 4,000 units at once on suspicion. If, in the meantime, we have been able to establish the diagnosis of laryngeal diphtheria we should repeat the dose at the end of twelve hours if the case is no worse, and twice this dosage if stenosis is progressing. A third injection is, as a rule, unnecessary and it may not even become necessary to intubate. This I have seen repeatedly.

In children under two years of age the dose should not exceed 3,000 units in the severer cases. In mild cases, which are seen early, 1,500 units is the maximum dose usually required. In order to obtain the best results, therefore, anti-

toxin must be used early and in sufficient potency to neutralize the toxins in the blood, repeating the dose if the action of the first one does not yield the desired results after a reasonable length of time.

The method of injection is simple. A site at which the skin is loose and not highly sensitive (preferably the axillary region or that of the shoulder-blade) is cleansed preparatory to the injection and the child is laid on its side. The injection is then made in the usual manner, and in order to avoid frightening the child unduly, we should keep the syringe out of sight and not make elaborate preparations before the unfortunate patient. Again, by injecting slowly, much unnecessary pain is avoided and loss of serum is likely to be less than by too hurried a procedure. Since the improvements in the preparation of serum have given us the same in a highly concentrated form, the necessity for employing an especially large syringe is done away with. Again, the sera on the market are put up in convenient packages, combining a container and aseptic syringe. However, it is well to have a special hypodermic syringe (one of 5 c.c. capacity) for this purpose, in order to insure of its being in readiness at all times. The needle should be boiled before inserting it under the skin, and the barrel of the syringe cleansed with an antiseptic solution before and after using. By carrying out a perfectly aseptic technique and employing a concentrated form of serum the local and general disturbances attributed to antitoxin will seldom be seen.

The beneficial effect of the serum upon the local and constitutional manifestations of the disease is noteworthy. First of all, there is a drop in the temperature. Repeated examinations of the throat will indicate that the membrane has ceased to spread, it becomes paler and cleaner in appearance, and at the end of twenty-four hours begins to loosen and shrivel. Within forty-eight hours an extensive membrane may have almost entirely disappeared, leaving behind only small fragments of the more firmly attached portions. Laryn-

geal stenosis is sometimes relieved sufficiently within a few hours to render intubation unnecessary. Nasal obstruction is relieved in a similar manner. As regards the constitutional symptoms, there is a rapid change from a condition of a most serious illness to a comparatively benign one. However, as said before, antitoxin does not prevent sequelæ, nor does it undo the mischief which has resulted from the action of the toxins upon the organs and tissues of the body. For this reason it is always wise to combine constitutional treatment with the antidotal treatment, with the object of counteracting these pathological processes and preventing sequelæ.

#### GLANDULAR FEVER.

This disease was first described by Pfeiffer (*Jahrbuch für Kinderheilk., Band. xxix., 1889*) and his observations have since been verified by both Continental and American observers.

The *etiology* is still obscure, but it is undoubtedly infectious, as it usually occurs in house-epidemics. West (*Archives of Pædiatrics*, Dec., 1896) reported an epidemic of ninety-six cases as having occurred in eastern Ohio. It is most frequently seen between the ages of two and eight, and in the fall and winter months (FILATOW).

The onset is abrupt, as a rule, the temperature reaching 103° F., or over. The child complains of loss of appetite, difficulty in swallowing, and pain on attempting to move the head from side to side. The bowels are constipated, and vomiting may be present. In conjunction with these symptoms there is coryza, injection of the mucous membrane of the throat, and slight difficulty in swallowing. The pathognomonic symptom is swelling of the glands of the neck, those situated just behind the point of origin of the sterno-cleido-mastoid muscle being most prominently enlarged. They are painful to the touch, and by careful palpation we can make out the nodular character of the swelling, pointing to the involvement of a series of individual glands. This



condition, in conjunction with the movability of the swelling, offers a ready distinction between glandular fever and mumps.

The fever may last for only a few days, or it may be prolonged to eight or ten days. In such case enlargement of the spleen and liver, scanty urine, and even albuminuria, may be observed. The swelling does not terminate in suppuration, but it may persist for some time after the fever has abated. I have seen it last for several weeks, with, however, gradual restoration to normal in every respect.

The *prognosis* is good. The *diagnosis* may require differentiation from *acute simple adenitis*, *mumps*, and *diphtheria*. The resemblance between the last two conditions is purely superficial, but they should be excluded in order to avoid error.

The chief remedies are *Belladonna* and *Mercurius*.

# INDEX

---

- Abscess, peritonsillar, 541**  
    retropharyngeal, 546  
    symptoms, 547 .  
    therapeutics, 548  
    treatment, 548
- Acetone in gastric contents, 142, 165**
- Acetonuria, 165**
- Acid hydrochloric, in gastric con-  
    tents, 143**
- Acidometer, 143**
- Adenitis, acute, 591**  
    tuberculous, 591  
        acute, 591  
        cervical, 591  
        chronic, 591  
        local, 591  
        mesenteric, 592  
        therapeutics, 592  
        tracheo-bronchial, 591  
        treatment, 592  
        surgical, 594
- Adenoid vegetations, 559**  
    diagnosis, 562  
    pathology, 560  
    symptomatology, 561  
    therapeutics, 565  
    treatment, 564  
        surgical, 565
- Airing, 12**
- Albumen water, 106**
- Albuminuria, cyclic, 365**  
    prognosis, 366  
    therapeutics, 367  
    treatment, 366  
    idiopathic, 365  
    orthostatic, 365
- Alcohol baths, 109**  
    indications for use of, 70
- Allenbury's foods, 110**
- Amœbic dysentery, 222**
- Anæmia, 422**  
    blood in, 424  
    pernicious, 426  
        blood in, 427  
        diet in, 427  
        etiology, 426  
        symptomatology, 426  
        therapeutics, 428  
        treatment, 427  
    pseudo-leukæmic, 432  
    secondary, 423  
    simple, 423  
        therapeutics, 428  
        treatment, 427  
    splenic (v. leukæmia), 423, 431  
    symptomatic, 423  
        therapeutics, 429  
    therapeutics, 428  
    with leucocytosis, 424
- Analysis of cerebro-spinal fluid, 462**  
    of gastric contents, 142  
    in cyclic vomiting, 165  
    in marasmus, 626  
    of milk, cow's, 83  
        human, 80  
    of stools, 188  
    of urine, 66
- Angina pectoris, 362**
- Ankle clonus, 48**
- Anomalies of heart (v. heart anom-  
    alies), 338**
- Antitoxin, diphtheria, 750**  
    dosage, 752  
    statistics, 752  
    technique of injection, 754  
    tetanus, 116
- Aortic regurgitation, 358**  
    stenosis, 358
- Aphthæ, Bednar's, 130**  
    epizooticæ, 130

- Aphthous stomatitis (v. stomatitis), 129  
 Apoplexy in newborn, 114  
 Appendicitis, 236  
     diagnosis, 238  
     symptomatology, 237  
     therapeutics, 239  
     treatment, 238  
     varieties, 237  
 Arteritis, umbilical, 115  
 Arthritis deformans, 612  
 Arthritism, 569  
 Artificial foods, 109  
 Asphyxia, extra-uterine, 112  
     intra-uterine, 112  
     neonatorum, 112  
     treatment, 113  
     sudden death from, 123  
 Asses' milk (v. milk), 79  
 Astasia abasia, 506  
 Asthma, 267  
     diagnosis, 269  
     of Millar, 254  
     symptomatology, 268  
     therapeutics, 269  
     thymic, 254  
     treatment, 269  
     varieties, 267  
 Atavism, 41  
 Ataxia, family, 519  
     diagnosis, 521  
     prognosis, 521  
     symptomatology, 520  
     hereditary cerebellar, 520  
 Atelectasis in newborn, 123  
 Athrepsia (v. marasmus), 160, 624  
 Atresia of stomach, 169  
 Atrophy of liver, acute yellow, 179  
 Aura, epileptic, 480  
 Auscultation, general methods, 59  
     in diseases of stomach, 140  
  
 Babinski's sign, 48, 439  
 Bacteria in milk, 100  
     of intestinal tract, 193  
 Baked flour, 108  
 Barley water in modified milk, 94, 105  
 Barlow's disease (v. scurvy), 584  
 Bathing, 9  
 Baths, alcohol, 109  
     bran, 18  
     cold, 18  
     hot, 18  
 Bednar's aphthæ, 130  
 Beef juice, 95, 106  
     tea, 106  
 Black measles, 637  
 Blood corpuscles, morphology, 418  
     diseases of, 418  
     erythrocytes, 418  
         determination, 421  
     examination, 421  
     in amœbic dysentery, 223  
     in anæmia, 424  
     in chlorosis, 425  
     in leukæmia, 431  
     in malaria, 694  
     in meningitis, cerebro-spinal, 690  
     in pernicious anæmia, 427  
     in pneumonia, 288  
     in rheumatism, 607, 612  
     in rickets, 580  
     in rubeola, 640  
     in typhoid fever, 708  
     leucocytes, 419  
         determination, 422  
         differential count, 422  
     pressure in childhood, 335  
     specific gravity, 418  
         determination, 422  
 Boils (v. furunculosis), 405  
 Bowels, regulation, 12  
 Brain, diseases of, 450  
 Brandy, use of, 109  
 Bright's disease (v. nephritis, chronic), 373  
 Broadbent's sign, 47  
 Bronchiectasis, 262, 265  
 Bronchitis, acute, 259  
     diagnosis, 262  
     pathology, 260  
     symptomatology, 261

- Bronchitis, therapeutics, 263  
     treatment, 262  
     varieties, 260  
 capillary, 262  
 chronic, 264  
     pathology, 264  
     symptomatology, 265  
     therapeutics, 265  
     treatment, 265  
 Broncho-pneumonia, acute, 270  
     diagnosis, 278  
     etiology, 270  
     pathology, 272  
     prognosis, 277  
     symptomatology, 275  
     therapeutics, 279  
     treatment, 278  
 Broths, 106  
 Buhl's disease, 116  
 Calculi, biliary, 179  
     renal, 382  
         symptomatology, 382  
         therapeutics, 383  
         treatment, 383  
     vesical (v. cystitis), 384  
 Camphor, uses of, 71  
 Cancer of stomach, 176  
 Cane-sugar in modifying milk, 88, 90  
 Carpo-pedal spasm, 490  
 Case, methods of taking, 41  
 Catarrh, gastric, acute, 152  
     gastric, chronic, 158  
     gastro-intestinal, chronic, 223  
     intestinal, acute, 209  
 Cephalalgia (v. headache), 527  
 Cephalæmatoma, 113  
 Cerebral palsy (v. palsy), 510  
 Cerebro-spinal fever, epidemic, 684  
     fluid, analysis of, 462  
     meningitis, epidemic, 684  
 Chapin dipper, 91  
 Chapin's method of modifying cow's  
     milk, 83, 91  
 Charcot-Leyden crystals, 191  
 Chicken broth, 106  
 Chicken-pox (v. varicella), 665  
 Childhood, periods of, 30  
 Chlorosis, 424  
     blood in, 425  
     etiology, 424  
     prognosis, 425  
     symptomatology, 424  
     therapeutics, 428  
     treatment, 427  
 Cholelithiasis, 179  
 Cholera infantum, 204  
     diagnosis, 207  
     etiology, 204  
     pathology, 205  
     symptomatology, 205  
     therapeutics, 218  
     treatment, 214  
 Chondrodystrophia foetalis, 572  
 Chorea, 492  
     cardiac, 498  
     diagnosis, 498  
     diet, 499  
     etiology, 492  
     in rheumatism, 612  
     laryngeal, 495  
     paralytic, 496  
     pathology, 494  
     post-hemiplegic, 495  
     prognosis, 498  
     symptomatology, 495  
     therapeutics, 499  
     treatment, 499  
 Chvostek's symptom, 490  
 Cirrhosis of liver, 180  
 Clinical examination, methods, 30  
 Clothing, 10  
 Club-foot, 520  
 Codliver oil, 109  
 Cold, therapeutics of, 17  
 Colic, 138  
     renal, 382  
 Colitis, follicular, 212  
     ileo-, acute, 209  
     membranous, 210, 213  
     diagnosis, 213  
 Colles' law, 617  
 Colostrum, 80, 98  
 Condensed milk, 110

- Constipation, 231  
   symptomatology, 231  
   therapeutics, 233  
   treatment, 232  
 Constitutional diseases, 567  
   remedies, 75  
 Consumption (v. tuberculosis), 297  
   galloping, 298  
 Contraction of stomach, 170  
 Convulsions (v. eclampsia), 473  
   epileptic (v. epilepsy), 478  
 Convulsive affections, 473  
 Costiveness, 231  
 Cowpox (v. vaccinia), 661  
 Cow's milk (v. milk, cow's), 83  
 Coxalgia, 506  
 Craniotabes, 46  
 Cremometer, 81  
 Cretinism, 448  
   sporadic, 449  
 Croup, membranous, 726, 733  
   spasmodic (v. laryngitis, acute catarrhal), 256  
 Cyclic albuminuria (v. albuminuria), 365  
   vomiting, 164  
 Cystitis, 384  
   symptomatology, 385  
   therapeutics, 386  
   treatment, 386  
   varieties, 385  
  
 Dactylitis, 620  
 Deaf-mutism, 448  
 Deformities of heart, 337  
 Dementia, 444  
 Dentition, 124  
   therapeutics, 126  
   treatment, 126  
 Development, in infancy, 32  
   muscular, in infants, 36  
 Diabetes insipidus, 378  
   diagnosis, 378  
   etiology, 378  
   pathology, 378  
   prognosis, 378  
   symptomatology, 378  
  
 Diabetes, therapeutics, 379  
   treatment, 379  
   mellitus, 379  
     diagnosis, 380  
     diet, 380  
     pathology, 380  
     symptomatology, 380  
     therapeutics, 381  
     treatment, 380  
 Diacetic acid in gastric contents, 142, 165  
 Diacetonuria, 165  
 Diarrhœa, acute infectious, 201  
   etiology, 202  
   diet, 203  
   chronic, 223  
     diagnosis, 225  
     pathology, 224  
     prognosis, 225  
     symptomatology, 224  
     therapeutics, 226  
     treatment, 226  
   fermental, 201, 207  
   simple (v. indigestion, acute intestinal), 196  
   therapeutics, 218  
 Diastase, 107  
 Diathesis, types of, 38  
 Diazo-reaction, 714  
 Diet (v. feeding), 77  
   at various periods, 96  
   in albuminuria, 366  
   in anæmia, 427  
   in cholera infantum, 216  
   in chorea, 499  
   in constipation, 232  
   in cystitis, 386  
   in diabetes mellitus, 380  
   in diarrhœa, acute, 216  
     chronic, 226  
   in epilepsy, 482  
   in gastralgia, 168  
   in gastritis, acute, 155  
     chronic, 161  
   in hepatic diseases, 181  
   in ileo-colitis, 216

- Diet, in indigestion, gastric, 147  
     intestinal, 198  
 in lithæmia, 570  
 in marasmus, 630  
 in nephritis, acute, 372  
     chronic, 377  
 in renal calculi, 383  
 in rheumatism, 613  
 in rickets, 582  
 in scarlatina, 652  
 in scrofula, 592  
 in scurvy, 586  
 in tuberculosis, intestinal, 230  
     pulmonary, 312  
 in typhoid fever, 716  
 in vomiting, cyclic, 166  
 Digestion in infancy, 183  
 Digitalin, use of, 71  
 Dilatation of stomach, 170  
 Diphtheria, 725  
     antitoxin, 750  
     diagnosis, 738  
     diet, 739  
     etiology, 725  
     intubation in, 740  
     laryngeal, 733  
     treatment, 740  
     nasal, 732  
     treatment, 740  
     paralysis after, 749  
     pathology, 727  
     prognosis, 736  
     pseudo-, 725, 734  
     clinical course, 735  
     scarlatinal, 736  
     symptomatology, 628  
     therapeutics, 745  
     tracheotomy in, 740, 744  
     treatment, 739  
 Diphtheroid, 725, 734  
 Diplegia, 513  
 Diseases, acute infectious, 635  
     of blood, 418  
     brain, 450  
     ear, 530  
     heart, 332  
     intestines, 183  
     Diseases of kidneys, 364  
     liver, 177  
     meninges, 450  
     mouth, 124  
     nervous system, 438  
     new-born, 31, 112  
     nose, 530  
     peritonæum, 248  
     respiratory tract, 254  
     skin, 393  
     stomach, 137  
     throat, 530  
     urinary organs, 364  
 Dosage of remedies, 74  
 Dysentery, 209  
     amoebic, 222  
     pathology, 209  
     symptomatology, 211  
     therapeutics, 222  
     treatment, 214  
 Dyspepsia (v. gastric indigestion,  
     acute), 146  
     nervous (v. gastric indigestion,  
     chronic), 149  
 Dystrophy, idiopathic muscular, 517  
     facio-scapulo-humeral, 518  
     infantile, 518  
     juvenile, 517  
     pathology, 517  
     peroneal, 518  
     therapeutics, 519  
     treatment, 518  
 Ear diseases, 530  
 Eclampsia, 473  
     diagnosis, 475  
     prognosis, 475  
     symptomatology, 474  
     therapeutics, 476  
     treatment, 476  
 Ectocardia, 338  
 Eczema, 394  
     definition, 394  
     diagnosis, 397  
     erythematous, 394  
     etiology, 395  
     intertrigo, 394

**Eczema, papulosum, 394**

pathology, 397

prognosis, 399

pustulosum, 394

squamosum, 395

symptomatology, 394

therapeutics, 400

treatment, 399

vesiculosum, 394.

**Effleurage, 29**

**Eggnog, 109**

**Eggs, 95**

**Emphysema, 318**

symptomatology, 319

therapeutics, 319

treatment, 319

**Empyema (v. pleurisy), 319**

**Endocarditis, 346**

foetal, 338

in rheumatism, 610

malignant, 350

symptomatology, 348

therapeutics, 351

treatment, 350

ulcerative, 350

**Enemata, 27**

**Enteroclysis, 28**

**Enteroptosis, 170**

**Enuresis, 387**

prognosis, 388

symptomatology, 387

therapeutics, 389

treatment, 388

**Epilepsy, 478**

diagnosis, 481

etiology, 478

hystero-, 504

Jacksonian, 474

prognosis, 481

symptomatology, 479

therapeutics, 483

treatment, 482

**Epiphysitis, 618**

**Erysipelas, new-born, 115**

**Erythema, 403**

caloricum, 404

definition, 403

**Erythema, intertrigo, 404**

medicamentosum, 405

scarlatinoides, 404

simplex, 403

therapeutics, 405

toxic, 403

traumaticum, 404

treatment, 405

venenatum, 404

**Exanthemata, 635**

**Exercise, 13**

**Fæces (v. stools), 184**

**Family ataxia (v. ataxia), 519**

**Farinaceous food, 94**

**Fats as food, 109**

**Fatty degeneration, acute, 116**

**Feeding (v. diet), 77**

adjuvant foods, 105

artificial foods, 105

forced (v. gavage), 26

infant, 77

intervals, 98

quantity of food, 98

time, 11

variation in foods, 97

**Fever, cerebro-spinal, 684**

glandular (v. glandular fever), 755

malarial (v. malaria), 694

spotted, 684

typhoid (v. typhoid fever), 700

**Flour, baked, 108**

**Foetal endocarditis, 338**

**Fontanels, closure of, 36**

**Formulae for modifying cow's milk,  
93**

**Friedreich's disease (v. ataxia), 519**

**Fruit juices, 109**

**Functional heart diseases (v. heart  
diseases), 362**

**Furunculosis, 405**

diagnosis, 406

symptomatology, 406

therapeutics, 407

treatment, 407

**Gallstones, 179**



- Gastralgia**, 167, 527  
     diagnosis, 167  
     etiology, 167  
     in rheumatism, 612  
     symptomatology, 167  
     therapeutics, 168  
     treatment, 168  
**Gastric contents, analysis of**, 142  
     spasm, congenital, 173  
     ulcer, 175  
**Gastritis, acute**, 151  
     afebrile, 153  
     catarrhal, 152  
     corrosive, 152  
     diagnosis, 155  
     etiology, 151  
     febrile, 153  
     follicular, 153  
     membranous, 153  
     pathology, 152  
     prognosis, 155  
     symptomatology, 153  
     therapeutics, 156  
     treatment, 155  
     chronic, 158  
         atrophic, 160  
         diagnosis, 161  
         etiology, 158  
         mucous, 160  
         pathology, 159  
         prognosis, 161  
         simple, 160  
         symptomatology, 159  
         therapeutics, 162  
         treatment, 161  
**Gastro-enteric intoxication, acute**, 207  
     diagnosis, 208  
     prognosis, 208  
     therapeutics, 218  
     treatment, 214  
**Gastro-intestinal catarrh, chronic**, 223  
**Gavage**, 15, 17, 26  
**German measles (v. rubella)**, 656  
**Glandular fever**, 755  
     diagnosis, 756  
     etiology, 755  
     prognosis, 756  
     symptomatology, 755  
     therapeutics, 756  
     treatment, 756  
**Globus hystericus**, 504  
**Glottis, spasm of**, 254  
     therapeutics, 255  
**Glycosuria**, 380  
**Gonorrhœa (v. vulvo-vaginitis)**, 390  
**Gonorrhœa, newborn**, 121  
**Grand mal**, 478  
**Grape juice**, 95  
**Grippe (v. influenza)**, 678  
**Growth in infancy**, 32  
  
**Hæmatoma of sterno-mastoid muscle**, 113  
**Hæmaturia**, 367  
     in scurvy, 585  
**Hæmoglobin, determination**, 421  
**Hæmoglobinuria**, 367  
     acute, in newborn, 117  
**Hæmophilia**, 433  
     pathology, 434  
     prognosis, 434  
     therapeutics, 435  
     treatment, 435  
**Hæmoptysis**, 306  
**Hæmorrhage, gastro-intestinal, new born**, 120  
     intracranial, newborn, 114  
**Hæmorrhagic diathesis (v. hæmophilia)**, 433  
**Hammer-toe**, 520  
**Headache**, 527  
     diagnosis, 528  
     etiology, 528  
     symptomatology, 528  
     therapeutics, 529  
     treatment, 528  
**Head-nodding (v. spasmodic nutans)**, 501  
**Heart anomalies, congenital**, 338  
     diagnosis, 341  
     symptomatology, 340  
     therapeutics, 342  
     treatment, 342

- Heart, defect of septum, 339  
 congenital, 337  
 deformities, 337  
 diseases of, 332  
 disease, chronic valvular, 354  
 functional, 362  
 murmurs, 336  
 patent ductus arteriosus, 339  
 patent foramen ovale, 339  
 stenosis of pulmonary artery, 339  
 symptomatology, 362  
 therapeutics, 363  
 treatment, 363  
 valvular defects, 340
- Heat, therapeutics of, 17
- Hemicrania, in rheumatism, 612
- Henoch's purpura, 436
- Hepatitis, 182
- Hernia, incarcerated, 235  
 strangulated, 235
- History, family, 41  
 of case, taking of, 41
- Hives (v. urticaria), 409
- Hodgkin's disease (v. leukæmia), 432
- Human milk (v. milk), 77
- Hutchinson's teeth, 620
- Hydrocephaloid, 206  
 therapeutics, 222
- Hydrocephalus, 468  
 acute (v. meningitis, tubercular), 453, 468  
 chronic, 468  
 diagnosis, 470  
 external, 468  
 ex vacuo, 468  
 internal, 468  
 symptomatology, 469  
 therapeutics, 472  
 treatment, 471
- Hydrotherapy, in typhoid, 717
- Hygiene, 9
- Hyperchlorhydria, 151
- Hyperostosis tibialis, 620
- Hypertrophy, tonsils (v. tonsils, hypertrophy), 543
- Hysteria, 502  
 accidents, 504  
 diagnosis, 508  
 prognosis, 508  
 stigmata, 503  
 symptomatology, 503  
 therapeutics, 509  
 treatment, 509
- Hystero-epilepsy, 504
- Icterus, catarrhal, 178  
 neonatorum, 119  
 physiological, 119
- Idiocy, 445  
 by deprivation, 448  
 cretinoid, 449  
 eclamptic, 446  
 epileptic, 447  
 genetous, 445  
 hydrocephalic, 446  
 inflammatory, 447  
 microcephalic, 446  
 paralytic, 447  
 sclerotic, 447  
 syphilitic, 447  
 therapeutics, 450  
 traumatic, 448  
 treatment, 449
- Ileo-colitis, acute (v. dysentery), 209  
 catarrhal, 211
- Imbecility, 445
- Impetigo contagiosa, 408  
 definition, 408  
 diagnosis, 409  
 etiology, 409  
 prognosis, 409  
 symptomatology, 408  
 therapeutics, 409  
 treatment, 409
- simplex, 407  
 definition, 407  
 diagnosis, 408  
 etiology, 408  
 prognosis, 408  
 symptomatology, 407  
 treatment, 408
- Impurities in milk, 83

- Inanition, acute, 628  
 Incubators, 14, 16  
 Indicanuria, 483  
 Indigestion, acute gastric, 146  
     etiology, 146  
     symptomatology, 147  
     therapeutics, 149  
     treatment, 147  
   acute intestinal, 196  
     diagnosis, 198  
     etiology, 196  
     symptomatology, 197  
     therapeutics, 199  
     treatment, 198  
   chronic gastric, 149  
     symptomatology, 150  
   infantile, 151  
 Infancy, development in, 32  
   growth in, 32  
   periods of, 30  
 Infantile convulsions (v. eclampsia),  
     473  
     paralysis (v. poliomyelitis), 515  
 Infantilism, 447  
 Infant feeding (v. feeding), 77  
   morbidity, 30  
   mortality, 31  
 Infants, delicate, 14  
   premature, 14  
 Infectious diseases, acute, 635  
 Influenza, 678  
   abdominal, 680  
   catarrhal, 680  
   cerebral, 680  
   diagnosis, 681  
   etiology, 679  
   neuralgic, 680  
   prognosis, 681  
   symptomatology, 679  
   therapeutics, 682  
   thoracic, 680  
   treatment, 682  
 Inhalation of steam, 21  
 Injection, rectal, 27  
 Insanity, 441  
   circular, 443  
   delusional, 443  
   epileptic, 442  
     hysterical, 443  
     masturbation, 444  
     morbid fears, 444  
     moral, 442  
     periodic, 443  
     progressive systematized, 443  
 Inspection, general methods, 44  
   in diseases of stomach, 139  
 Interstitial nephritis (v. nephritis),  
     376  
 Intertrigo, 404  
 Intestinal catarrh, acute (v. dysen-  
     tery), 209  
   obstruction, 234  
   parasites, 241  
   tuberculosis, 227  
 Intestines, diseases of, 183  
 Intubation, 740  
   diphtheria, 740  
   nasal, 744  
 Intussusception, 235  
 Inunctions, 28  
   nutritive, 28  
 Irrigation of colon, 27  
 Ischæmia, 423  
 Itch (v. scabies), 416  
  
 Jaundice, catarrhal, 178  
   new-born (v. icterus), 119  
 Junket, 106  
  
 Kernig's sign, 49, 439, 688  
 Kidneys, acute degeneration, 366  
   diseases of, 364  
 Knee-jerk, 48, 439  
 Koplik's sign, 636  
 Kyphosis, 578  
  
 Lactic acid in gastric contents, 144  
   test for, 144  
 Lactobutyrometer, 82  
 Lactometer, 81  
 Lactose in modified milk, 88, 90  
 La grippe (v. influenza), 678  
 Laryngismus stridulus, 254  
 Laryngitis, acute catarrhal, 256

- Laryngitis**, diagnosis, 257  
     etiology, 256  
     symptomatology, 256  
     therapeutics, 258  
     treatment, 258  
**Lavage**, 21, 148  
     contra-indications, 26  
     in chronic gastritis, 162  
     method of performing, 24  
**Leptomeningitis** (v. meningitis), 450  
**Leucocytosis with anæmia**, 424  
**Leukæmia**, 431  
     pseudo-, 432  
     symptomatology, 431  
     therapeutics, 433  
     treatment, 433  
**Lice** (v. pediculosis), 415  
**Liebig's food**, 107  
**Lithæmia**, 567  
     etiology, 567  
     symptomatology, 568  
     therapeutics, 571  
     treatment, 570  
**Lithuria**, 569  
**Liver**, cirrhosis, 180  
     diseases of, 177  
         therapeutics, 181  
         treatment, 181  
     examination of, 177  
     yellow atrophy, 179  
**Lumbar puncture**, 458  
     diagnostic value, 462  
     in meningitis epidemic, 692  
     operative technique, 460  
**Lymphatism**, 122, 586  
  
**Macewen's sign**, 53  
**Malaria**, 694  
     blood in, 694  
     diagnosis, 698  
     etiology, 694  
     irregular, 697  
     masked, 697  
     pathology, 695  
     prognosis, 697  
     symptomatology, 695  
  
**Malaria**, therapeutics, 698  
     treatment, 698  
**Malarial cachexia**, 697  
     fever, 694  
**Malformations of stomach**, 169  
**Malnutrition**, 624, 629  
     diagnosis, 629  
**Malpositions of stomach**, 169  
**Malt diastase**, 107  
**Malted milk**, Horlick's, 110  
**Malt extract**, 108  
**Maltine**, 108  
**Mania**, 442  
**Marasmus**, 624  
     analysis of gastric contents in, 626  
     diagnosis, 629  
     diet, 630  
     etiology, 626  
     prognosis, 629  
     symptomatology, 627  
     therapeutics, 633  
     treatment, 629  
**Massage**, 28  
**Mastitis in newborn**, 119  
**Masturbation insanity**, 444  
**Measles** (v. rubeola), 635  
     German (v. rubeola), 656  
**Melancholia**, 443  
**Melena**, 120  
**Mellin's food**, 110  
**Membranous croup**, 726, 733  
**Meninges**, diseases of, 450  
**Meningitis**, 450  
     basilar (v. meningitis, tuberculous), 453  
     cerebro-spinal, epidemic, 450, 684  
         abortive, 687  
         complications, 690  
         diagnosis, 690  
         etiology, 684  
         fulminating, 686  
         pathology, 685  
         prognosis, 690  
         protracted, 687  
         sequelæ, 690  
         symptomatology, 686  
         temperature in, 689

- Meningitis, therapeutics, 692**  
     treatment, 691  
     varieties, 686  
**leptomeningitis, acute, 450**  
     diagnosis, 453  
     pathology, 451  
     prognosis, 452  
     symptomatology, 451  
     therapeutics, 465  
     treatment, 464  
**posterior basic, 454, 691**  
**pseudo-, 507**  
**tuberculous, 453**  
     diagnosis, 458  
     pathology, 454  
     prognosis, 457  
     symptomatology, 455  
     therapeutics, 467  
     treatment, 464  
**Methods of clinical examination, 30**  
     of prescribing, 72  
**Microscopical examination of human milk, 82**  
     of stools, 190  
**Migraine, 528**  
**Milk and cream mixtures, 92**  
     asses' milk compared with human, 79  
     cows', analysis of, 83  
         bacteria in, 83  
         compared with human, 78  
         condensed, 110  
         digestion of, 183  
         "Ideal" testers, 84  
         impurities in, 83  
         malted, 110  
         modification of, 86  
             barley water in, 94, 105  
             cane-sugar in, 88, 90  
             diluent in, 90, 94, 105  
             formulae, 93  
             lactose in, 88, 90  
             milk and cream mixtures, 92  
             top milk method, 90  
         peptonized, 107  
         preservatives in, 83  
         top milk, 83, 90  
**Milk, human, analysis of, 80**  
     bacteria in, 100  
     compared with cows', 78  
     composition, 77  
     microscopical examination, 82  
     strippings, 84  
     variations in, 84  
     infection, acute (v. cholera infantum), 204  
     sugar in milk modification, 88, 90  
**Mitral regurgitation, 356**  
     stenosis, 357  
**Modification of milk, 86**  
**Morbidity, infant, 30**  
**Morbilli (v. rubeola), 635**  
**Morbus maculosus Werlhofii, 436**  
**Mortality, infant, 31**  
**Motor affections, 492**  
**Mouth, care of, 10**  
     diseases of, 124  
     putrid sore, 131  
**Mucous disease (v. diarrhoea), 223**  
**Multiple neuritis (v. neuritis), 524**  
     sclerosis (v. sclerosis), 523  
**Mumps (v. parotitis), 675**  
**Muscular development in infants, 36**  
     dystrophy (v. dystrophy), 517  
**Mutton broth, 106**  
**Mycotic disease (v. thrush), 31, 132**  
**Myocarditis, 352**  
     diagnosis, 353  
     prognosis, 354  
     symptomatology, 353  
     therapeutics, 354  
     treatment, 354  
**Nephritis, acute, 368**  
     diet, 372  
     etiology, 369  
     pathology, 369  
     prognosis, 371  
     symptomatology, 370  
     therapeutics, 372  
     treatment, 371  
     varieties, 368  
     chronic, 373  
         interstitial, 376

- Nephritis, pathology, 376**  
     prognosis, 376  
     therapeutics, 377  
     treatment, 377  
     parenchymatous, 374  
     pathology, 374  
     prognosis, 375  
     symptomatology, 374  
     therapeutics, 377  
     treatment, 377  
**Nervous system, diseases of, 438**  
**Neuralgia, 526**  
     diagnosis, 527  
     therapeutics, 527  
     treatment, 527  
**Neuritis, multiple, 524**  
     diagnosis, 525  
     symptomatology, 525  
     therapeutics, 526  
     treatment, 526  
**Newborn, apoplexy in, 114**  
     asphyxia, 112  
         sudden death from, 123  
     atelectasis, 123  
     care of, 9  
     diseases of, 31, 112  
     erysipelas, 115  
         therapeutics, 115  
         treatment, 115  
     fatty degeneration, acute, 116  
     gastro-intestinal hæmorrhage, 120  
         therapeutics, 121  
     gonorrhœa, 121  
     hæmoglobinuria, 117  
     icterus, 119  
     intracranial hæmorrhage, 114  
     mastitis, 119  
         therapeutics, 119  
         treatment, 119  
     œdema, 120  
     omphalitis, 115  
     ophthalmia (v. ophthalmia), 117  
     peritonitis, 115  
     sepsis in, 114  
         pathology, 114  
     sudden death, 122  
     tetanus, 115  
     Newborn, umbilical arteritis, 115  
         phlebitis, 115  
     Night terrors, 444  
     Noma (v. stomatitis), 134  
     Nose, diseases of, 530  
         syringing, 19  
     Nurse, wet, 86  
     Nursing, 9  
     Nystagmus, 501  
  
**Oatmeal water, 106**  
**Objective symptoms, 72**  
**Obstruction, intestinal, acute, 234**  
     diagnosis, 236  
**Œdema, newborn, 120**  
     without kidney lesion, 367  
**Oligochromæmia, 432**  
**Oligocythæmia, 432**  
**Omphalitis, newborn, 115**  
**Ophthalmia neonatorum, 117**  
     prognosis, 118  
     treatment, 118  
**Orange juice, 95**  
**Organic heart disease, 354**  
**Orthostatic albuminuria, 365**  
**Osteochondritis, 620**  
**Osteogenesis imperfecta, 572**  
**Otitis, 530**  
     influenzal, 532  
     in scarlatina, 647  
     media, acute catarrhal, 532  
         diagnosis, 535  
         prognosis, 534  
         purulent, 532  
         symptomatology, 533  
         therapeutics, 535, 537  
         treatment, 535  
     tubercular, 532  
**Oxaluria, 385**  
**Ozæna, 554**  
  
**Pack, cold, 19**  
     hot, 19  
     mustard, 19  
**Palpation, general methods, 50**  
     in diseases of stomach, 141  
**Palsy, cerebral, 510**

- Palsy, prognosis, 513  
     symptomatology, 512  
 Paracentesis, 327  
 Paralysis, post-diphtheritic, 749  
     pseudo-hypertrophic, 518  
     spinal, 515  
 Paralytic affections, 510  
 Paranoia, 443  
 Paraplegia, hereditary spastic, 521  
     pathology, 521  
     symptomatology, 521  
 Parasites in stools, 192  
 Parasites, intestinal, 241  
     morphology, 243  
     therapeutics, 245  
     treatment, 244  
     varieties, 241  
 Parasitic diseases, animal, 415  
     vegetable, 411  
 Parenchymatous nephritis (v. nephritis), 374  
 Parotitis, 675  
     diagnosis, 677  
     epidemic, 675  
     prognosis, 677  
     secondary, 676  
     symptomatology, 676  
     therapeutics, 678  
     treatment, 678  
 Pasteurization, 102  
 Pasteurizer, Freeman's, 102  
 Pectus carinatum, 47, 578  
 Pediculosis capitis, 415  
     definition, 415  
     diagnosis, 415  
     symptomatology, 415  
     treatment, 415  
 Peliosis rheumatica (v. purpura), 436  
 Pemphigus, 409  
 Pepsin, test for, 145  
 Peptogenic milk powder, 107  
 Peptonized food in prematurity, 15  
     milk, 107  
 Percentage of food constituents, 97  
 Percussion, abdomen, 58  
     chest, 54  
     Percussion, factors influencing, 55  
         general methods, 53  
         head, 53  
         in stomach diseases, 140  
 Pericarditis, 342  
     in rheumatism, 611  
     pathology, 342  
     prognosis, 345  
     symptomatology, 343  
     therapeutics, 345  
     treatment, 345  
 Periodic vomiting, 164  
 Periods, of childhood, 30  
     of infancy, 30  
 Peritonæum, diseases of, 248  
 Peritonitis, acute, 248  
     pathology, 248  
     symptomatology, 249  
     therapeutics, 249  
     treatment, 249  
     chronic (v. tuberculous peritonitis), 251  
     new-born, 115  
     tuberculous, 251  
         diagnosis, 252  
         prognosis, 252  
         symptomatology, 251  
         therapeutics, 253  
         treatment, 253  
 Peritonsillar abscess, 541  
 Pertussis, 667  
     complications, 671  
     diagnosis, 672  
     etiology, 667  
     pathology, 668  
     prognosis, 671  
     sequelæ, 671  
     symptomatology, 668  
     therapeutics, 673  
     treatment, 672  
 Petit mal, 478  
 Petrissage, 29  
 Phimosis, 387  
 Phlebitis, umbilical, 115  
 Phthisis, chronic fibroid, 308  
     pneumonic, 298  
 Physical diagnosis, 44



- Pial hæmorrhage, 112  
 Pityriasis linguæ, 129  
 Pleurisy, 319  
     diagnosis, 324  
     pathology, 320  
     physical signs, 322  
     prognosis, 325  
     symptomatology, 321  
     therapeutics, 329  
     treatment, 326  
         operative, 328  
 Pleuro-pneumonia (v. pneumonia), 295  
 Plica polonica, 415  
 Pneumonia, abortive, 290  
     broncho-, acute, 270  
     catarrhal, 270  
     central, 289  
     cerebral, 288  
     croupous (v. lobar), 283  
     influenzal, 290  
     lobar, 283  
         complications, 290  
         diagnosis, 294  
         etiology, 283  
         pathology, 284  
         physical signs, 291  
         prognosis, 293  
         symptomatology, 285  
         therapeutics, 296  
         treatment, 296  
         varieties, 288  
     lobular, 270  
     pleuro-, 295  
         therapeutics, 296  
         treatment, 296  
     typhoid, 290  
     wandering, 289  
 Polioencephalitis, acute 511  
 Poliomyelitis, acute, anterior, 515  
     diagnosis, 516  
     pathology, 515  
     prognosis, 516  
     symptomatology, 515  
 Polyuria, 376, 378, 380, 507  
 Prematurity, 14  
     diet in, 15  
     Prescribing, methods of, 72  
     Preservatives in cow's milk, 83  
     Profeta's law, 618  
     Pseudo-diphtheria, 725, 734  
     Pseudo-hypertrophic paralysis, 518  
     Pseudo-leukæmia, 423, 432  
     Pseudo-meningitis, 507  
     Ptyalin, test for, 145  
     Pulmonary tuberculosis (v. tuberculosis), 297  
     Pulse in childhood, 63, 335  
     Pump, stomach, 22  
     Purpura, 435  
         fulminans, 437  
         hæmorrhagica, 436  
         Henoch's, 436  
         rheumatica, 436  
         simplex, 435  
         therapeutics, 437  
         treatment, 437  
     Pyuria, 385  
  
     Quantity of food in infancy, 98  
     Quinsy (v. tonsillitis, parenchymatous), 541  
  
     Rachitic rosary, 577  
     Rachitis, 572  
         acute, 581  
         diagnosis, 581  
         etiology, 572  
         foetal, 572  
         Pasteurized milk in, 104  
         pathology, 574  
         prognosis, 581  
         symptomatology, 576  
         teeth in, 128  
         therapeutics, 582  
         treatment, 582  
     Râles, 63  
     Record keeping, 41  
     Reflexes, 48  
     Regurgitation, aortic, 358  
         mitral, 356  
     Remedies, constitutional, 73  
     Renal calculi (v. calculi), 382  
     Renal colic, 382

- Respiration in childhood, 63  
     puerile type, 62  
 Respiratory tract, diseases of, 254  
 Retropharyngeal abscess (v. abscess), 546  
 Rheumatic fever, 606  
 Rheumatism, 606  
     anæmia in, 612  
     articular, 606, 610  
     chorea in, 612  
     chronic, 612  
     diet, 613  
     endocarditis in, 610  
     etiology, 607  
     gastralgia in, 612  
     hemicrania in, 612  
     muscular, 611  
     pericarditis in, 611  
     symptomatology, 609  
     therapeutics, 613  
     tonsillitis in, 537, 611  
     treatment, 613  
 Rhinitis, acute, 548  
     symptomatology, 549  
     therapeutics, 551  
     treatment, 550  
     atrophic, 554  
         etiology, 554  
         prognosis, 556  
         symptomatology, 555  
         therapeutics, 558  
         treatment, 557  
     chronic, simple, 552  
         symptomatology, 553  
         therapeutics, 558  
         treatment, 556  
     hypertrophic, 553  
         etiology, 554  
         prognosis, 556  
         symptomatology, 555  
         therapeutics, 558  
         treatment, 556  
     pseudo-membranous, 550  
         therapeutics, 552  
         treatment, 551  
     purulent (v. chronic), 552  
 Rice water, 105  
     paste, 105  
 Rickets (v. rachitis), 572  
 Ringworm (v. tinea circinata), 413  
 Rötheln (v. rubella), 656  
 Rubella, 656  
     diagnosis, 657  
     etiology, 656  
     morbilliforme, 656  
     prognosis, 657  
     scarlatiniforme, 656  
     symptomatology, 656  
     therapeutics, 657  
     treatment, 657  
     varieties, 656  
 Rubeola, 635  
     blood in, 640  
     complications, 640  
     desquamation, 639  
     etiology, 635  
     symptomatology, 636  
     temperature in, 639  
     therapeutics, 641  
     treatment, 640  
 Rupture (v. hernia), 235  
 Sabre-blade deformity, 620  
 Saint Vitus' dance (v. chorea), 492  
 Scabies, 416  
     definition, 416  
     diagnosis, 416  
     etiology, 416  
     pathology, 416  
     prognosis, 416  
     therapeutics, 417  
     treatment, 416  
 Scarlatina, 643  
     complications, 655  
     desquamation, 648  
     diagnosis, 650  
     diet, 652  
     etiology, 644  
     lævigata, 646  
     miliaris, 646  
     prognosis, 649  
     sequelæ, 655  
     symptomatology, 645

- Scarlatina, temperature in, 646  
     therapeutics, 652  
     treatment, 650  
     variagata, 646  
 Scarlet fever (v. scarlatina), 643  
 Sclerosis, disseminated, 523  
     multiple, 523  
         diagnosis, 524  
         symptomatology, 523  
         therapeutics, 524  
         treatment, 524  
 Scorbutus (v. scurvy), 584  
 Scrofula, 587  
     diagnosis, 590  
     erethetic, 589  
     etiology, 588  
     phlegmatic, 589  
     prognosis, 590  
     symptomatology, 589  
     therapeutics, 592  
     treatment, 592  
 Scurvy, 584  
     diagnosis, 585  
     etiology, 584  
     infantile, 584  
     Pasteurized milk in, 104  
     symptomatology, 585  
     therapeutics, 586  
     treatment, 586  
 Seat-worms, 243  
 Secondary anæmia, 423  
 Sepsis in new-born, 114  
 Serum therapy, 750  
 Shiga's bacillus, 195, 202  
 Simple anæmia, 423  
 Skin, diseases of, 393  
 Sleep, 11  
 Small-pox (v. variola), 658  
 Snuffles, 619  
 Sore mouth, putrid, 131  
 Spasm, congenital gastric, 173  
     glottis, 254  
         prognosis, 254  
         symptomatology, 254  
         treatment, 255  
 Spasmus nutans, 501  
     prognosis, 502  
     treatment, 502  
 Spastic paraplegia, 521  
 Splenic anæmia (v. leukæmia), 431  
 Sponging, cold, 13  
 Spotted fever, 450, 684  
 Spraying, throat, 20  
 Status choreicus, 498  
     lymphaticus, 122, 586  
 Stenosis, aortic, 358  
     mitral, 357  
     pyloric, 170  
     stomach, 169  
 Sterilization of food, 101  
 Sterilizer, Arnold steam, 102  
 Stethoscope, binaural, 60  
     monaural, 60  
 Stiller's phenomenon, 47  
 Still's disease, 612  
 Stimulants, use of, 70, 109  
 Stomach, atresia, 169  
     cancer, 176  
     contraction of, 170  
     dilatation, 170  
         diagnosis, 173  
         etiology, 171  
         symptomatology, 173  
         treatment, 174  
     diseases of, 137  
         absorption of food in, 145  
         auscultation in, 140  
         etiology, 137  
         inspection in, 139  
         mensuration in, 140  
         motility of, 145  
         palpation in, 141  
         percussion in, 140  
         vomiting in, 141  
     malformations, 169  
     malpositions, 169  
     stenosis, 169  
         pyloric, 170  
     tube, 21  
     ulcer, 175  
         follicular, 175  
         round perforating, 175  
         symptomatology, 176

- Stomach, ulcer, therapeutics, 176  
     treatment, 176  
     tuberculous, 175  
     washing out (v. lavage), 21  
 Stomatitis, aphthous, 129  
     etiology, 129  
     pathology, 130  
     symptomatology, 130  
     therapeutics, 135  
     treatment, 134  
 catarrhal, 128  
     etiology, 128  
     symptomatology, 128  
     therapeutics, 135  
     treatment, 134  
 gangrenous, 134  
     etiology, 134  
     pathology, 134  
     prognosis, 134  
     symptomatology, 134  
     therapeutics, 135  
     treatment, 134  
 parasitic, 132  
     diagnosis, 133  
     etiology, 132  
     pathology, 132  
     prognosis, 133  
     symptomatology, 133  
     therapeutics, 135  
     treatment, 134  
 ulcerative, 131  
     etiology, 131  
     pathology, 131  
     symptomatology, 132  
     therapeutics, 135  
     treatment, 134  
 Stone (v. calculi), 382  
 Stools, bloody, 187, 192  
     analysis of, 188  
     color, 185, 188  
     decrease in number, 187  
     frequent, 186  
     in cholera infantum, 206  
     in constipation, 231  
     in fermental diarrhoea, 208  
     in dysentery, 211  
     in gastro-intestinal catarrh, 224  
     in indigestion, acute, 197  
     in intestinal tuberculosis, 229  
     in marasmus, 628  
     large, 185  
     liquid, 186  
     microscopical examination, 190  
     mucous, 187  
     normal infantile, 184  
     parasites in, 192  
     pus in, 192  
 Stridor, congenital, 254  
 Strippings, 84  
 Strychnia, use of, 71  
 Subjective symptoms, 72  
 Sudden death in infants, 122  
 Symptomatic nervous affections, 526  
 Symptoms, objective, 72  
     subjective, 72  
 Syphilis, 617  
     acquired, 617  
     congenital, 617  
     germinal, 617  
     hereditary, 617  
     diagnosis, 621  
     pathology, 618  
     prognosis, 621  
     symptomatology, 619  
     therapeutics, 622  
     treatment, 622  
 Syringing, nasal, 19  
 Syringomyelia, 521  
     diagnosis, 522  
     prognosis, 522  
     symptomatology, 522  
  
 Tabes mesenterica, 228  
 Taché cérébrale, 45, 53, 456, 507  
 Talipes equinus, 516  
 Tape-worms, 242  
 Teeth, abnormalities, 127  
     care of, 10  
     Hutchinson, 127  
     in rachitis, 128  
 Temperament, 38  
 Temperature in rubeola, 639  
     in scarlatina, 646  
     normal, 66

- Test-meal, 143  
 Tetanus, 115  
   antitoxin, 116  
   therapeutics, 116  
   treatment, 116  
 Tetany, 489  
   diagnosis, 491  
   etiology, 489  
   prognosis, 491  
   symptomatology, 490  
   therapeutics, 491  
   treatment, 491  
 Tetter (v. eczema), 394  
 Therapeutics, general methods, 69  
 Throat, diseases of, 530  
   spraying, 20  
 Thrush, 31, 132  
 Thymic death, 122  
 Tinea, 411  
   circinata, 413  
     diagnosis, 414  
     etiology, 414  
     prognosis, 414  
     symptomatology, 414  
     therapeutics, 414  
     treatment, 414  
   tonsurans, 412  
     definition, 412  
     diagnosis, 412  
     etiology, 412  
     pathology, 412  
     prognosis, 412  
     symptomatology, 412  
     therapeutics, 413  
     treatment, 413  
 Tonsillitis, acute, 537  
   cryptic (v. folliculous), 538  
   folliculous, acute, 538  
     diagnosis, 539  
     symptomatology, 539  
     therapeutics, 540  
     treatment, 540  
   parenchymatous, acute, 541  
     prognosis, 542  
     symptomatology, 542  
     therapeutics, 543  
     treatment, 542  
   rheumatic, 537, 611  
     superficial, acute, 538  
     symptomatology, 538  
   ulcero-membranous, 541  
     therapeutics, 541  
     treatment, 541  
 Tonsillotomy, operative technique, 545  
 Tonsils, hypertrophy, 543  
   etiology, 544  
   symptomatology, 544  
   therapeutics, 545  
   tonsillotomy in, 545  
   treatment, 544  
 Top milk, 83, 90  
 Torticollis, 611  
 Tracheotomy, in diphtheria, 740, 744  
 Trousseau's symptom, 490  
 Tuberculin, 303  
 Tuberculosis, 595  
   chronic, 602  
   congenital, 597  
   diagnosis, 604  
   diet in, 605  
   etiology, 595  
   intestinal, 227  
     pathology, 228  
     prognosis, 229  
     symptomatology, 229  
     therapeutics, 230  
     treatment, 230  
   latent, 597  
   miliary, 297  
     acute, 601  
   pathology, 598  
   protracted, 602  
   pulmonary, caseous, 298  
     diagnosis, 301  
     pathology, 298  
     prognosis, 300  
     symptomatology, 299  
     therapeutics, 302  
     treatment, 303  
   chronic, 303  
     diagnosis, 309  
     diet, 312  
     prognosis, 308

- Tuberculosis, symptomatology, 305  
     therapeutics, 312  
     treatment, 310  
     fibro-caseous, 303  
     symptomatology, 600  
     therapeutics, 604  
     treatment, 604  
 Tuberculous adenitis (v. adenitis),  
     587, 591  
     diathesis, 595  
     meningitis (v. meningitis), 453  
     peritonitis (v. peritonitis), 251  
 Typhoid fever, 700  
     abortive, 709  
     blood in, 708  
     complications, 709, 712  
     diagnosis, 712  
     diet, 716  
     eruption, 707  
     etiology, 700  
     hydrotherapy, 717  
     lesions, 700  
     pathology, 703  
     pneumonia, 290  
     prognosis, 711  
     pulse, 708  
     relapses, 709  
     septicæmic, 704  
     symptomatology, 704  
     temperature, 704, 706, 711  
     therapeutics, 720  
     treatment, 716  
     urine in, 708  
  
 Uffelmann's test, 144  
 Ulcer of stomach, 175  
 Uræmia, 375  
 Uric acid diathesis (v. lithæmia), 567  
 Urinary tract, diseases of, 364  
 Urine, blood in, 367  
     in cholera infantum, 206  
     in cyclic vomiting, 165  
     in cystitis, 385  
     in diabetes insipidus, 378  
     mellitus, 579  
     in diagnosis, 66  
     in epilepsy, 483  
  
 Urine, in hysteria, 507  
     in kidney disease, 364  
     in lithæmia, 569  
     in nephritis, acute, 370  
         chronic interstitial, 376  
         parenchymatous, 374  
     in scurvy, 585  
     in tuberculosis, pulmonary, 308  
     in typhoid fever, 708  
     in yellow atrophy of liver, 179  
 Urticaria, 409  
     diagnosis, 410  
     etiology, 410  
     papulosa, 410  
     pigmentosa, 410  
     prognosis, 411  
     symptomatology, 410  
     therapeutics, 411  
     treatment, 411  
  
 Vaccination, 662  
     operative technique, 662  
 Vaccine lymph, 663  
 Vaccinia, 661  
     prognosis, 664  
     symptomatology, 663  
     therapeutics, 665  
     treatment, 665  
 Vaginitis (v. vulvo-vaginitis), 390  
 Valvular heart disease, chronic, 354  
     prognosis, 356  
     stages, 355  
     symptomatology, 354  
     therapeutics, 360  
     treatment, 359  
 Varicella, 665  
     diagnosis, 666  
     etiology, 665  
     gangrænosa, 666  
     symptomatology, 665  
     therapeutics, 667  
     treatment, 667  
 Variola, 658  
     diagnosis, 660  
     etiology, 658  
     pathology, 658  
     prognosis, 660

Variola, symptomatology, 658  
therapeutics, 661  
treatment, 660

Varioloid, 660

Ventilation, 12

Vernix caseosa, 9

Vesical calculi (v. cystitis), 384

Volvulus, 235

Vomiting, cyclic, 142, 164  
prognosis, 166  
symptomatology, 165  
therapeutics, 166  
treatment, 166  
in stomach diseases, 141  
periodic, 164

Vulvo-vaginitis, 390  
non-specific, 390  
specific, 391  
therapeutics, 392  
treatment, 392

Weaning, 95

Weight chart, 36  
curve, 36

Wet nurse, 86

Whooping cough (v. pertussis), 667

Widal's test, 713

Winkel's disease (v. hæmoglobi-  
nuria), 117

Worms (v. parasites), 241













LANE MEDICAL LIBRARY

STANFORD UNIVERSITY

This book should be returned on or before  
the date last stamped below.

25M-3-58 00267

P45 Raue, Charles  
R24 Diseases of children.  
1906 2d ed.

[illegible]



